Science-Based Solutions

Canadian Tissue Residue Guidelines for the Protection of Wildlife Consumers of Aquatic Biota: Methylmercury

Report No. 1-4

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Canadian Tissue Residue Guidelines for the Protection of Consumers of Aquatic Life: Methylmercury

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This particular issue provides the scientific supporting information and rationale for the development of Canadian Tissue Residue Guidelines for the protection of consumers of aquatic life for methylmercury. The information in this document is current as of 2000, when the document was originally prepared. Minor revisions and editorial changes have been made for publication in 2002. For additional information regarding these guidelines, please contact:

Environment Canada
Environmental Quality Branch
National Guidelines and Standards Office
351 Saint-Joseph Boulevard
Hull, QC K1A 0H3

phone: 819-953-1550
fax: 819-953-0461
ceqg-rcqe@ec.gc.ca
http://www.ec.gc.ca

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CCME Documents
c/o Manitoba Statutory Publications
200 Vaughan Street
Winnipeg, MB R3C 1T5

tollfree: 800-805-3025
fax: 204-945-7172
spccme@gov.mb.ca
http://www.ccme.ca

This scientific supporting document is available in English and in French. A factsheet is also available in English under the title *Canadian Environmental Quality Guidelines* (CCME 1999). Ce document scientifique du soutien est disponible en français et en anglais. Un feuillet d'information est aussi disponible en français sous le *titre Recommandations canadiennes pour la qualité de l'environnement* (CCME 1999).

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ABSTRACT

This document reviews the background information and rationale for the derivation of the Canadian Tissue Residue Guideline (TRG) for methylmercury (MeHg) for the protection of wildlife consumers of aquatic biota. It outlines information on the physical and chemical properties of MeHg, the sources and uses of mercury, production of MeHg, environmental fate and behaviour, bioaccumulation in biota, levels of MeHg in Canadian biota and various environmental media, and describes the toxicological effects of MeHg in mammals and birds. This abstract will briefly outline the information to be found in each chapter of the report. Tables are located either within the text or following each chapter, and all figures and Appendices are at the end of the report following the References.

> To protect Canadian wildlife that consume fish or shellfish from any toxicological effects of MeHg, aquatic biota should contain no more than **33 $\mu g \cdot kg^{-1}$** (0.033 $mg \cdot kg^{-1}$) MeHg on a wet weight basis.

The derivation and rationale for the wet-weight based TRG are fully described in chapter 10. It is important to note that the TRG was derived using the reference concentration (RC) of the Wilson's storm petrel (susceptible to MeHg bioaccumulation because it consumes almost its entire body weight in aquatic biota per day). In regions where this species is not found, RC values for resident wildlife may be applied (Table 12). The use of these other RC values will result in allowable concentrations that are higher than the TRG. **Monitoring programs in Canada typically measure mercury concentrations in large sport fish that generally contain higher levels than small fish (<35 cm), which are consumed by wildlife.** As such, mercury levels reported herein are biased toward those for large fish and may not represent actual risks to wildlife.

Mercury can exist in many forms, both inorganic and organic. Elemental mercury (Hg^0) is very volatile, and is the only metal that is liquid at room temperature. The most toxicologically relevant form of mercury is MeHg (CH_3Hg). Methylmercury is produced through the biological and chemical methylation of inorganic mercury. It is a potent neurotoxicant for animals and humans. Methylmercury is not very lipid soluble compared to many organochlorine contaminants, but it binds strongly with sulphydryl groups in proteins and is therefore readily accumulated and retained in biological tissues (Clarkson 1994).

There are many sources of mercury to the environment, both natural and human related. Natural sources include geological mercury deposits, forest fires and other wood burning activities, volcanoes, and volatilisation from the oceans. Humans may accelerate the natural weathering process of mercury by removing it from areas rich in geological deposit and introducing or re-introducing it to the mercury cycle. Consumer and industrial products, coal and other fossil fuel combustion, chlorine alkali processing, mercury waste in landfills or storage, waste incineration, cement manufacture, metal smelting, and other activities have all altered the natural Hg cycle.

While world production (mining) and use of mercury is declining, substantial quantities are still used in some countries for gold mining. Mercury has been used in dental amalgams, pesticides, thermometers, barometers, and electrical products such as dry-cell batteries, fluorescent lights, switches, and other control equipment. It is also used in substantial quantities

in the electrolytic preparation of chlorine and caustic soda (chlor-alkali industry) (AEP 1992).

Generally, the aquatic mercury cycle may be characterised by high atmospheric fallout, removal by deposition to sediments, re-emission to the atmosphere as elemental Hg (Hg^0), methylation and demethylation transformation processes within the lake or watershed, and exchanges through tributaries and groundwater (runoff and seepage) (Hurley et al. 1994). Some portion of the total mercury (THg) pool is accumulated by biota.

Methylmercury has a high potential for bioaccumulation as well as biomagnification with increasing trophic levels. Mercury and MeHg may be taken up by aquatic organisms directly from water or through their diet. Uptake by animals directly from water is the result of dissolved mercury adsorption or absorption through the body surface and respiratory organs. Available data indicate that MeHg usually represents less than 50% of total mercury in freshwater aquatic plants. The proportion of MeHg (MeHg/THg) increases through the food web, being lowest in aquatic plants, intermediate in invertebrates, and highest in fish and birds.

Fish, and piscivorous mammals and birds accumulate most of their mercury body burdens through their diet. Mercury in fish is comprised almost exclusively of MeHg regardless of the composition of diet sources and exposure water. Methylmercury levels in wildlife consumers of fish are elevated relative to the fish or other aquatic biota they consume indicating its biomagnification potential. Selenium (Se) is known to reduce the accumulation of mercury and to exert a measure of protection against MeHg toxicity. The Hg-Se ratio has shown a tendency to be higher in freshwater fish than in marine fish, which may be an indication of the higher relative mercury levels in certain freshwater environments (Pelletier 1985).

With respect to animal uptake, about 95% of an ingested dose of MeHg is efficiently absorbed into the bloodstream from the gastrointestinal tract, whereas inorganic Hg is less readily absorbed (7 to 15%; Wolfe et al. 1998). Methylmercury distributes via the bloodstream to all parts of the body, readily crosses placental barriers, may enter the foetal brain, and is readily accumulated in growing animal fur, bird feathers or human hair. The accumulation of mercury in the brain is greater in mammals than in fish (Zillioux et al. 1993). Some early clinical signs of MeHg toxicity in mammals include constricted vision, loss of co-ordination and nervous tremors. Birds often suffer reproductive impairment without an indication of obvious neurological effects.

Many countries have mercury or MeHg guidelines to protect human consumers of fish and shellfish. Only the United States and Canada, however, are in the process of establishing guidelines to protect wildlife that consume fish and other aquatic biota. The US EPA "Mercury Report to Congress" is a series of comprehensive reviews of all aspects of mercury and MeHg in the environment, including the derivation of water concentrations and tolerable consumption levels to protect wildlife consumers of fish (US EPA 1997b,c).

RÉSUMÉ

Le présent document expose les informations et les critères qui ont servi à élaborer la Recommandation canadienne visant les résidus dans les tissus (RRT) pour le méthylmercure (MeHg), dont l'objectif est de protéger les espèces fauniques qui consomment des organismes aquatiques. Il présente dans ses grandes lignes l'information sur les propriétés chimiques et physiques du MeHg, les sources et les utilisations du mercure, la production du MeHg, le devenir et le comportement du mercure dans l'environnement ainsi que sa bioaccumulation dans les biotes, et les concentrations de MeHg dans le biote canadien et divers compartiments de l'environnement; il décrit aussi les effets toxiques du MeHg chez les mammifères et les oiseaux. Le présent résumé décrit brièvement l'information présentée dans chaque chapitre du rapport. Certains tableaux ont été placés au fil du texte et d'autres, à la fin des chapitres; toutes les figures et annexes se trouvent à la fin du rapport, après la section des références bibliographiques.

Afin de protéger les espèces fauniques canadiennes qu consomment des poissons ou des crustacés et coquillage contre tout effet toxique du MeHg, les organismes aquatique ne devraient pas contenir plus de **33 µg·kg^{-1}** (0,033 mg·kg^{-1}) d MeHg (poids frais).

La méthode et les critères qui ont servi à élaborer la RRT basée sur le poids frais sont décrits en détail au chapitre 10. Il est important de remarquer que la RRT a été établie à partir de la concentration de référence (CR) de l'Océanite de Wilson (oiseau particulièrement susceptible de bioaccumuler le MeHg, étant donné qu'il consomme quotidiennement presque l'équivalent de son poids en organismes aquatiques). Dans les régions où cette espèce n'est pas présente, les CR des espèces résidentes peuvent être appliquées (tableau 12). L'utilisation de ces autres CR entraînera des concentrations admissibles plus élevées que la RRT. **Au Canada, les programmes de surveillance mesurent généralement les concentrations de mercure dans les grands poissons de sport, qui contiennent habituellement des concentrations plus élevées que les petits poissons (<35 cm) consommés par la faune.** Ainsi, les concentrations de mercure mentionnées dans le présent document correspondent plutôt à celles mesurées chez les grands poissons et peuvent n'être pas représentatives des risques réels pour la faune.

Le mercure peut exister sous plusieurs formes, tant inorganiques qu'organiques. Le mercure élémentaire (Hg^o) est très volatil et est aussi le seul métal liquide à la température de la pièce. La forme de mercure la plus importante sur le plan toxicologique est le MeHg (CH_3Hg). Le méthylmercure est produit par méthylation biologique et chimique du mercure inorganique. Il constitue un neurotoxique puissant pour les animaux et les humains. Le méthylmercure n'est pas très liposoluble par rapport à de nombreux contaminants organochlorés, mais il se lie solidement aux groupes sulfhydryles des protéines et est donc facilement accumulé et retenu dans les tissus biologiques (Clarkson, 1994).

Le mercure présent dans l'environnement provient de nombreuses sources naturelles et anthropiques. Les sources naturelles comprennent les gisements mercurifères, les feux de forêts et toute autre combustion de bois, les volcans et la volatilisation du mercure présent dans les océans. Les humains peuvent accélérer le processus naturel de mobilisation du mercure sous l'effet de l'altération météorique des roches mercurifères, en le déplaçant des régions riches en gisements géologiques et en l'introduisant ou en le réintroduisant dans le cycle du mercure. Certains produits de consommation et de l'industrie, la combustion du charbon et d'autres combustibles fossiles, la production de chlore et de soude caustique, les déchets de mercure dans les décharges ou les entrepôts, l'incinération des déchets, les cimenteries et les fonderies sont au nombre des facteurs qui ont altéré le cycle naturel du mercure.

Bien que la production (exploitation minière) et l'utilisation mondiales du mercure diminuent, des quantités importantes sont toujours utilisées dans certains pays dans l'exploitation de l'or. Le mercure a été utilisé dans les amalgames dentaires, les pesticides, les thermomètres, les baromètres et divers produits électriques tels que les piles sèches, les tubes fluorescents, les interrupteurs et d'autres dispositifs de contrôle. Le mercure est également utilisé en quantités importantes dans la production électrolytique de chlore et d'hydroxyde de sodium (soude caustique) (AEP, 1992).

De façon générale, le cycle du mercure dans le milieu aquatique peut être caractérisé de la façon suivante : dépôt atmosphérique important, dépôt dans les sédiments, réémission dans l'atmosphère sous forme de mercure élémentaire (Hg^o), méthylation et déméthylation dans le lac ou le bassin hydrographique, et transport dans les cours d'eau et les eaux souterraines (ruissellement et infiltration) (Hurley *et al.*, 1994). Une partie du mercure total (HgT) s'accumule dans le biote.

Le méthylmercure a un important potentiel de bioaccumulation, ainsi que de bioamplification d'un niveau trophique à l'autre. Le mercure et le MeHg peuvent être absorbés par les organismes aquatiques directement de l'eau, ou par leur alimentation. Dans le premier cas, le mercure dissous est adsorbé ou absorbé au niveau de la surface corporelle et des organes respiratoires. Les données disponibles indiquent que le MeHg représente habituellement moins de 50 % du mercure total présent dans les plantes aquatiques d'eau douce. La proportion de MeHg (MeHg/HgT) augmente d'un niveau à l'autre du réseau trophique : elle est au plus bas chez les plantes aquatiques, intermédiaire chez les invertébrés et au plus haut chez les poissons et les oiseaux.

Les poissons, les mammifères piscivores et les oiseaux accumulent la plus grande partie de leur charge corporelle de mercure par leur alimentation. Le mercure présent dans les poissons est presque exclusivement sous forme de MeHg, quelles que soient la composition du régime alimentaire des poissons et de l'eau dans laquelle ils se trouvent exposés au mercure. Les concentrations de méthylmercure dans les animaux piscivores sont élevées par rapport à celles mesurées chez les poissons ou d'autres organismes aquatiques qu'ils consomment, ce qui témoigne de son potentiel de bioamplification. On sait que le sélénium (Se) réduit l'accumulation du mercure et offre une protection contre la toxicité du MeHg. Le ratio Hg:Se tend à être plus élevé chez les poissons dulcicoles que chez les poissons marins, ce qui pourrait indiquer des concentrations relatives de mercure plus élevées dans certains milieux d'eau douce (Pelletier, 1985).

En ce qui a trait à l'absorption du mercure par les animaux, environ 95 % d'une dose ingérée de MeHg est efficacement absorbée par le tractus gastro-intestinal et se retrouve dans le sang, alors que le Hg inorganique est moins facilement absorbé (7 % à 15 %; Wolfe *et al.*, 1998). Le méthylmercure est distribué dans toutes les parties du corps par le sang, traverse facilement les barrières placentaires, peut pénétrer le cerveau du fœtus, et s'accumule facilement dans la fourrure, les plumes ou les cheveux humains durant leur croissance. L'accumulation de mercure dans le cerveau est plus importante chez les mammifères que chez les oiseaux (Zillioux *et al.*, 103-00500/

1993). Certains des premiers signes cliniques de l'intoxication par le MeHg chez les mammifères comprennent une vision réduite, une perte de la coordination et des tremblements nerveux. Chez les oiseaux, on observe souvent des problèmes de reproduction, sans signes évidents de troubles neurologiques.

De nombreux pays ont des lignes directrices pour le mercure ou le MeHg qui visent à protéger les personnes qui consomment du poisson et des coquillages et crustacés. Cependant, seuls les États-Unis et le Canada sont en train d'élaborer des lignes directrices visant à protéger les espèces fauniques qui consomment des poissons ou d'autres organismes aquatiques. Le « Mercury Report to Congress » de l'EPA des États-Unis présente des examens détaillés de tous les aspects du mercure et du MeHg dans l'environnement, dont les méthodes d'établissement des concentrations dans l'eau et des concentrations tolérables dans le biote permettant d'assurer la protection des animaux piscivores (US EPA, 1997b,c).

1.0 INTRODUCTION

Methylmercury (CH_3HgX, hereafter abbreviated as MeHg) is one of many forms of mercury that may be found in natural environments. It is approximately ten times more toxic than inorganic mercury to biota and is a potent neurotoxicant. Methylmercury is the form of mercury that is most readily accumulated by biota, and is only slowly excreted because of strong binding with proteins (Clarkson 1994).

As a mercury species, MeHg is not produced by humans, but rather it is produced a) biologically by micro-organisms, and b) chemically in natural aquatic environments under certain circumstances. Inorganic mercury occurs naturally in rocks all over the world. Humans cause an accelerated weathering of inorganic mercury when they extract it from the earth for their use, and may extend its life in the global mercury cycle when they re-use or incinerate mercury-containing products. Since MeHg is produced from inorganic mercury, high MeHg concentrations may be found where inorganic mercury occurs. Excessive inorganic mercury is released to the environment where humans use it during mining, ore smelting, fossil fuel and solid waste incineration, chlor-alkali plants (OECD 1994), and other uses that will be described in this report. Once released to the atmosphere, mercury may be carried hundreds or thousands of miles to be deposited in watersheds and surface waters. It is under certain aquatic conditions that MeHg is produced from the deposited or *in situ* inorganic mercury, and enters the food web.

Levels of mercury are higher in natural environments now and in recent history than in pre-industrial times. This has been demonstrated by changes in mercury flux rates in lake sediment cores collected in North America and Europe (e.g., Lucotte et al. 1995; Engstrom and Swain 1997; Landers et al. 1998). Recognising the environmental and health-related problems that mercury may cause, many governments have placed restrictions on human fish consumption because of mercury contamination, have banned or are phasing out the use of mercury in consumer and industrial products, and are curtailing industrial emissions of mercury (WHO 1990; OECD 1994; Environment Canada and US EPA 1997; US EPA 1997b,c).

Fish monitoring programs have shown that piscivorous fish may have elevated MeHg in their tissues. Likewise, avian and mammalian wildlife that are dependent on aquatic biota such as fish or shellfish are susceptible to accumulating toxic levels of MeHg (reviewed by Wolfe et al. 1998). This report will comprehensively examine the evidence that indicates that avian wildlife species exposed to excessive MeHg may incur reproductive and behavioural deficits, while mammalian piscivores are more likely to suffer neurological effects. Based on this evidence, a Canadian Tissue Residue Guideline (TRG) for MeHg was developed to protect piscivorous birds and mammals.

Development of the Canadian TRG for MeHg involved a review of the scientific literature on the production, uses, sources, and levels of MeHg in biota in Canada. The chemical properties, bioaccumulation, fate and behaviour, including the important processes of mercury methylation and demethylation were evaluated to characterise the types of sites in Canada most likely to develop MeHg contamination. Diet-based acute and chronic toxicity data for avian receptors such as loons, herons and eagles, and for piscivorous mammals such as mink and otter, were evaluated according to the protocol for deriving Canadian TRGs for the protection of wildlife consumers of aquatic biota (CCME 1998).

2.0 PHYSICAL AND CHEMICAL PROPERTIES

2.1 Properties

Mercury (Hg^o) belongs to group IIb of the periodic table of elements, and is chemically quite different from the other two members of the group, Zn and Cd. For example, of the three metals, only Hg may become methylated in the environment to form alkylmercury compounds [such as mono methylmercury (CH_3Hg^+) and dimethylmercury ($(CH_3)_2Hg$)] and aryl compounds (phenyl-mercury) (AEP 1992). The mechanism involves the nonenzymatic transfer of methyl groups (CH_3) from methylcobalamin (CH_3B_{12}) to Hg^{2+} (Robinson and Tuovinen 1984), a process that will be described in more detail in chapter 5:

$$Hg^{2+} \xrightarrow{CH_3B_{12}} \underset{\textit{Mono-MeHg}}{CH_3Hg^+} \xrightarrow{CH_3B_{12}} \underset{\textit{Di-MeHg}}{(CH_3)_2Hg}$$

Unlike the other metals, mercury has a strong tendency toward covalent interactions, and has a high chemical affinity for thiol (S-H) groups. Compared to cadmium or zinc, covalent complexes of Hg^{2+} are many orders of magnitude more stable. For example, the stability constants for $HgCl_2$, $CdCl_2$ and $ZnCl_2$ and are 10^{16}, 10^3 and 10^{-1}, respectively (AEP 1992). Unlike mercury, cadmium and zinc compounds are not volatile nor are they reduced or methylated by microorganisms.

Mercury has a very low melting point (-37°C) compared with cadmium (321°C) and zinc (420°C). Elemental mercury (Hg^o) is the only metal that is liquid at room temperature. It has a high surface tension, forming spherical droplets when the liquid is released. It is also of low solubility in water, making precipitation and aquatic systems a poor sink for Hg^o. Elemental Hg is volatile; with volatility increasing with temperature. Its high surface tension and uniform volume of expansion make mercury ideal for use in thermometers, barometers and other measuring devices (Smith and Rowan-West 1996).

Mercury can theoretically exist in various valence states (0, +1, and +2), and forms inorganic as well as organic compounds. The chemical compounds of Hg (II) are much more numerous than those of Hg (I) (OECD 1994). The inorganic forms include the very stable, and relatively water-soluble halides, $HgCl_2$, $HgBr_2$, and HgI_2, plus the water-soluble, dissociated form, HgF_2 (AEP 1992). Among the inorganic anions, Hg^{2+} forms the strongest covalent bond with chloride ions, so mercuric-chloride species may dominate when chloride salts are abundant (AEP 1992).

In addition to simple salts, such as chloride, nitrate and sulphate, mercury (II) forms an important class of organometallic compounds. These are characterised by the attachment of mercury to either one or two carbon atoms to form compounds of the type RHgX and RHgR' where R and R' represent the organic moiety (most commonly CH_3). The most numerous are those of the type RHgX, where X may be an anion such as chloride (Cl^-), sulphide, or sulphate (SO^-_4) (WHO 1989).

The most toxicologically relevant form of mercury is methylmercury (CH_3Hg). Hg is easily transported across cell membranes, resulting in toxicity to biota. Of the mercury compounds, methylmercury has the highest octanol-water partition coefficient ($K_{ow} = [Hg]_{octanol}:[Hg]_{water}$; log K_{ow} 1.7-2.5; Table 1), which is an indication of its affinity for body fats. While this log K_{ow} is moderate relative to many organochlorine substances (e.g., toxaphene 6.44; DDT compounds

>5.5), it is the combination of lipophilicity and especially its binding with sulphydryl groups in proteins which fosters retention of MeHg (Saouter et al. 1993).

2.2 Sample Collection and Analytical Methods

Until the last decade or so, many mercury measurements in water samples were artificially high because of sample contamination (Driscoll et al. 1994). For example, mercury concentrations in water from Vandercook Lake, Wisconsin, saw an apparent decrease from over 200 $ng \cdot L^{-1}$ in 1983 to ~50 $ng \cdot L^{-1}$ in 1985 to 0.5 $ng \cdot L^{-1}$ in 1986 as investigators used progressively cleaner techniques for sample collection and handling (Fitzgerald and Watras 1989). Coincidentally, formerly undetectable levels of mercury and MeHg (such as in filtered water samples) are now commonly reported because of improvements in analytical techniques. Notably, the detection limits for MeHg are often in the $pg \cdot L^{-1}$ range (Lee 1987; Bloom 1989). At the Third International Conference on "Mercury as a Global Pollutant" in July 1994, an entire session was devoted to mercury collection and measurement methods [see Vol. 80 (1-4), Water Air Soil Pollut. 1995].

2.2.1 Water sample collection

The use of trace-metal clean collection techniques is increasingly being recognised as necessary for samples used for mercury analyses. Typically, Teflon bottles are first washed with hot HNO_3, filled with a 1% ACS grade HCl solution, and stored in new sealable polyethylene bags (St. Louis et al. 1994). Bottles should be transported to the field within clean containers that may also be placed inside plastic bags. All handlers must wear new polyethylene gloves. At the site, the HCl solution is drained and the bottle rinsed three times with sample water, filled, and then placed back into the bags. If a sample pump is used, acid-rinsed Teflon tubing should be used throughout. Within 2 h of sample collection, a small quantity (250 µL per 250 mL water) of concentrated *trace metal* grade HCl is added as a preservative (St. Louis et al. 1994).

Unfiltered samples may then be used for mercury analysis. For dissolved mercury analyses, filtering should be done with either an acid-cleaned glass (Ramamoorthy 1982) or Teflon filtration apparatus using acid-cleaned polycarbonate filters (e.g., 0.4 µm nucleopore filters; Gill and Bruland 1990; Hurley et al. 1995).

2.2.2 Analysis of Total Mercury and Methylmercury in Water

The methods of analysis used worldwide for mercury and its compounds are too numerous to detail in this report, however, the recently developed and widely preferred method will be detailed. Descriptions of some other methods in use are tabulated from pages 248 to 255 in US DOH and HS (1994).

Bloom (1989) and Watras et al. (1995)[1] analysed for total mercury (THg), dissolved mercury, MeHg, plus Hg^{o}. They used a cryogenic gas chromatograph with a highly sensitive cold vapour atomic fluorescence detector (CVAFS). Methylmercury is determined by aqueous-phase ethylation followed by purging of volatile methyl ethyl mercury onto graphitized carbon black (Carbotrap). Hg^{o} is determined by sparging 500 mL of unfiltered, untreated water for 20 min with Hg-free N_2 and trapping the volatile Hg on gold. Methylmercury and Hg^{o} are both thermally desorbed from their respective "traps" prior to CVAFS detection. Detection limits range from

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[1] Both of these first authors were directly involved in developing US EPA Method 1631 (1996).

0.01 to 0.06 $ng\cdot L^{-1}$ for THg, from 0.01-0.02 $ng\cdot L^{-1}$ for MeHg, and from 0.004 to 0.02 $ng\cdot L^{-1}$ for Hg^{o} (Watras et al. 1995).

An excerpted summary of this recent standard technique developed by scientists for the US EPA (1996) follows. This method is increasingly being used in Canada, such as at the Freshwater Institute (DFO) in Winnipeg (St. Louis et al. 1994).

Summary of EPA Method 1631 (US EPA 1996).

A 100–2000 mL sample is collected directly into specially cleaned, pretested, fluoropolyme bottle(s) using sample handling techniques specially designed for collection of mercury a trace levels (US EPA 1995a).

For dissolved Hg, samples are filtered through a 0.45-µm capsule filter.

Samples are preserved by adding 5 $mL\cdot L^{-1}$ of pretested 12 N HCl (to allow both total an methyl Hg determination) or 5 $mL\cdot L^{-1}$ BrCl solution, if total mercury only is to be determined.

Prior to analysis, a 100 mL sample aliquot is placed in a specially designed purge vessel and 0.2 N BrCl solution is added to oxidize all Hg compounds to Hg(II).

After oxidation, the sample is sequentially prereduced with $NH_2OH\cdot HCl$ to destroy the fre halogens, and then reduced with $SnCl_2$ to convert Hg(II) to volatile Hg(0).

The Hg(0) is separated from solution by purging with nitrogen onto a gold-coated sand trap.

The trapped Hg is thermally desorbed from the gold trap into an inert gas stream that carrie the released Hg(0) into the cell of a cold-vapor atomic fluorescence spectrometer (CVAFS for detection.

2.2.3 Analysis of Methylmercury in Sediment and Tissue Samples

The analysis for tissue or sediment samples would be the same as for water samples except the sample is either 1) digested with a mixture of hot (180°C) nitric and sulphuric acids, or 2) treated with 60°C nitric and sulphuric acids and oxidized with a solution of potassium permanganate, with excess permanganate titrated with 30% peroxide, or 3) microwave digested in the presence of nitric acid. In all cases, the oxidized mercury is converted to the elemental state with a reducing solution containing stannous chloride (or stannous sulphate), hydroxylamine sulphate, and sodium chloride (Hendzel and Jamieson 1976; Scruton et al. 1994). The mercury is partitioned into vapour and determined by GC/CVAA or GC/CVAFS as described above.

A recent modification to these procedures is that methylmercury is first distilled from all matrix types into a Teflon vessel, ethylated with sodium tetraethyl borate and sorbed onto a Tenax (Enka Research Institute, Anaheim CA) resin column. Methylmercury is then separated using gas-liquid chromatography, pyrolized, and detected as Hg^{o} using CVAFS (Liang et al. 1994).

The Canadian Food Inspection Agency, which is responsible for monitoring mercury concentrations in fish for domestic use and for export, analyses tissue for total mercury with the assumption that essentially all mercury in fish tissue is methylmercury (E. Burns-Flett 1999, DFO, pers. com.).

Table 1. Physical and chemical properties of mercury.

Property[a]	**Mercuric Hg (II) ($HgCl_2$)**	**Methylmercury (CH_3Hg^+)**	**Elemental Mercury (Hg^o)**
Atomic number	--	--	80
Molecular Weight	271.5	215.63	200.59
Boiling point	--	$Hg(CH_3)_2$: 92°C/98.66 kPa (4)	356.9°C/ 101.32 kPa (1)
CAS No.	7487-94-7	22967-92-6	7439-97-6
Conductivity	--	--	$0.022\ cal \cdot s^{-1} \cdot cm^{-3} \cdot {}^oC^{-1}$ (1)
Density (spec. gravity)	5.4 (4)	$Hg(CH_3)_2$: 3.1874 (4)	$13.534\ g \cdot cm^{-3}$ at 25°C (4)
Melting point	--	--	38.87°C
K_{OC} (organic carbon)[b]	Not avail.	DOC increases solubility (6)	Not avail.
log K_{sed}	3.4-4.1 (3)	2.9-3.2 (reduced by DOC in water; 6)	--
log $K_{susp.part.}$	5.35 (2); 4.8-4.9 (10)	5.73(2)	--
log K_{ow}	0.61 (5)	1.7(7)-2.54(5)	--
Surface tension	--	--	$475\ dynes \cdot cm^{-1}$ (1)
Vapour pressure, Pa	$1.87 \cdot 10^{-2}$ @ 34°C (4)	1.13 @ 20°C MeHgCl (11)	0.16 @ 20°C (11) $2 \cdot 10^{-3}$ at 25°C (10)
Water Solubility	$69\ g \cdot L^{-1}$ (4)	$1\ \mu g \cdot L^{-1}$ (8);	$25\text{-}30\ \mu g \cdot L^{-1}$ (9)

Numbers in parentheses refer to the following references: (1) AEP 1992; (2) Hurley et al. 1989; (3) Hurley et al. 1994; (4) RSC 1994; (5) Halbach 1985; (6) Miskimmin 1991; (7) Major et al. 1991; (8) Eisler 1987; (9) Krenkel 1974; (10) Hurley et al. 1995; and (11) WHO 1990.

Notes:

[a] With the exception of molecular weight, most of these properties are dependent on pH, temperature, carbon content and other characteristics, and are given as general values only.

[b] K refers to a partition coefficient of a substance between indicated phase:water (Hg concentrations in each medium). For example, K_{ow}=octanol/water partition coefficient; $K_{susp.part.}$=suspended particle/water; $K_{sed.}$=sediment/water; K_{oc}=organic carbon/water.

3.0 PRODUCTION AND USES

3.1 Mining of Mercury

Mercury cannot be created or destroyed. Human activities introduce excess mercury into the global mercury cycle that would otherwise have remained buried. The mining of mercury essentially results in an accelerated weathering process, by which much more mercury than normal is released from rocks.

Relatively few countries are involved in the production of mercury from mining cinnabar or other deposits. Important international mining locations include Algeria, Russia, China, Finland, Spain, and Mexico (OECD 1994; Table 2). Canada has no mercury mines that are currently active. Two mines were formerly active in central British Columbia along the Pinchi Fault zone. The Pinchi Mine was operational in the early 1940s and briefly in the early 1970s, and the Bralorne Takla Mine was operational for one year in the 1940s (Armstrong 1946; A. Plouffe 1999, GSC, pers. com.).

Mercury production from mining has declined steadily in the last 20 years (OECD 1994). Worldwide production decreased from 10 000 t in 1970 to 5 500 t in 1990. There is significant secondary production of mercury, which includes recycling, recovery, and industrial reprocessing of mercury. In the United States, secondary industrial production varied between 10 and 20% of total mercury consumption in 1985-89 (Minerals Yearbook 1989).

From 1972 to 1984, Mexico was the main Hg supplier to the largest gold mining nation, Brazil who requires about 340 t of Hg per year mainly for its gold mining processes (Veiga et al. 1995). Worldwide, the total number of gold miners using mercury amalgamation to extract gold in the early 1990s was estimated to be 4 ± 1 million persons (Brazil 650 000; Tanzania 250 000; Indonesia 250 000; Vietnam 150 000) (Jernelöv and Ramel 1994).

Stockpiles of mercury in the United States are maintained for strategic defence purposes by the National Defense Stockpile (NDS) and the Department of Energy (DOE). NDS legislation enacted in 1992 authorised the disposal of the entire mercury stock from the NDS. At the end of 1993, there was an estimated 4466 t of mercury in the NDS groups (US Department of Commerce 1993).

3.2 Uses

Mercury has been used in dental amalgams, pesticides, thermometers, barometers, and electrical products such as dry-cell batteries, fluorescent lights, switches, and other control equipment. It is also used in substantial quantities in the electrolytic preparation of chlorine and caustic soda (chlor-alkali industry) (AEP 1992). Smaller quantities are used in industrial catalyst manufacture, general laboratory use, and pharmaceuticals. (US EPA Factsheet; www.epa.gov/OGWDW)

According to an Organisation for Economic Co-operation and Development (OECD) questionnaire submitted to member countries (including Canada) the general range of consumption patterns for all countries for 1988-92 were as follows:

- batteries, range 3 to 69% (average 25%);
- chlor-alkali industry, range 2 to 78% (average 28%);

- electrical equipment and measurement equipment, range 1 to 50% (average 16%);
- paint, range 0.1 to 16% (average 10%);
- tooth fillings, range 2 to 51% (average 7%);
- other uses, such as thermometers and laboratory uses (average 14%) (OECD 1994).

Fehr and Dennis (1975) estimated that 75% of seeds for cereal crops in Alberta were formerly treated with mercury compounds and that up to 120 t of organic mercury compounds were applied to Alberta and Saskatchewan soils from 1930 to 1972. During this period, seed-eating and predatory birds, as well as their eggs, were recognised to have elevated methylmercury concentrations that originated from the fungicide treatments of the seeds. Some of the mercury used in the 95 agricultural products that were registered for use may still be a component of the mercury cycle. By the early 1990s, there were four remaining mercurial fungicides registered for use in Canada, all of which were for turf application (Sang and Lourie 1997). Since December 31 1995, no further registration of mercury-containing fungicides was permitted although the remaining retail stocks could be used until depleted. These retail stocks were estimated to be depleted by the end of 1997.

According to the OECD survey, Canada used an average of 56.7 t Hg·a^{-1} from 1982 to 1991. Of this amount 24 t was reported to be used in measurement equipment, 10 t in dental amalgams, with approximately 4 t for each of batteries, thermometers, electrical switches, and in laboratory uses. Chlor-alkali industries, paints, and pesticides were each reported at approximately 2 t Hg·a^{-1} in Canada (OECD 1994; Table 3). A summary of Canadian mercury use by sector is also found in Pilgrim and EMAN (1998).

The remaining mercury cell chlor-alkali plants in Canada are regulated federally by the Chlor-Alkali Mercury Liquid Effluent Regulation[2] and the Chlor-Alkali Mercury Release Regulations[3] with respect to the release of mercury to the environment. Chlor-alkali plants produce chlorine and caustic soda (which is used by the pulp and paper industry) by passing a brine solution over mercury cells. A status report prepared by Environment Canada summarised the loss of mercury from mercury cell chlor-alkali plants to effluents, emissions, products, and solid wastes for the five mercury cell plants that were operating in Canada from 1986-1989. During 1986-1989, total mercury losses to effluent decreased from 88.07 kg·a^{-1} (1986) to 46.29 kg·a^{-1} (1989). Total mercury emissions over the four year period decreased from 680.56 kg·a^{-1} (1986) to 547.18 kg·a^{-1} (1989). Total mercury losses to products and solid wastes for all plants during 1986-1989 were 302.64 kg and 3318.13 kg, respectively (Paine 1994).

In the U.S., chlor-alkali plants used the most mercury (381 t), followed by batteries (250 t), paint (192 t), electrical switches (141 t) and measurement equipment (87 t; OECD 1994). These figures were as of 1989, and were reported in the 1995 OECD survey (Table 3).

Because mercury is an important long-range atmospheric pollutant, the decline in use of mercury by the U.S. (former world's largest Hg user; 1214 t used in 1989), may have important long term implications for mercury cycling in Canada. In the U.S., the use of mercury-containing products in 1990 was reduced by a factor of 3.6 compared to the maximum in 1968. One important reason for this decline is that mercury is no longer used in agriculture, amalgamation, the pulp and paper industry, and the pharmaceutical industry. Electrical equipment, batteries and the chlor-alkali industry each account for one third of consumption. Paint accounted for up to 18% of mercury consumption until 1990, when a decrease occurred following new restrictions on mercury use in paints. Battery use showed a substantial downward trend from 1989 to 1990, whereas other uses showed smaller changes since 1984 (OECD 1994).

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[2] Chlor-Alkali Mercury Liquid Effluent Regulations, C.R.C., c.811.

[3] Chlor-Alkali Mercury Release Regulations, SOR/90-130

3.2.1 Use Reduction

Mercury and its compounds are now ranked as Level I persistent toxic substances on a list prepared by Environment Canada and the United States Environmental Protection Agency, in a bilateral strategy to eventually eliminate anthropogenic toxic substances in the Great Lakes Basin (Environment Canada and US EPA 1997). As such, goals concerning reductions in the use of mercury compounds have been set by each country.

The U.S. goal is to seek by the year 2006, a 50% reduction nationally in the deliberate use of mercury and a 50% reduction in the release of mercury from sources resulting from human activity. The release challenge will apply to the aggregate of releases to the air nation-wide and of releases to the water within the Great Lakes Basin. This target is considered as an interim reduction target and, in consultation with stakeholders, will be revised if warranted, following circulation of the Mercury Study Report to Congress (US EPA 1997b,c).

The Canadian goal is to seek by the year 2000, a 90% reduction in the release of mercury, or where warranted, the use of mercury, from polluting sources resulting from human activity in the Great Lakes Basin. This target is considered as an interim reduction target and, in consultation with stakeholders in the Great Lakes Basin, will be revised if warranted, following completion of the 1997 COA (Canada-Ontario Agreement Respecting the Great Lakes Basin Ecosystem, established in 1994).

Activities by Canadian companies in the Great Lakes Basin to date have resulted in significant reductions in mercury content in batteries (60 – 90%), fluorescent lamps (44%) and switches, while further reductions are planned, such as 70% by fluorescent lamp manufacturers by 2000 (Environment Canada and US EPA 1997). Other Canadian strategies are summarised in Appendix A.

Table 2. Worldwide production of mercury from ores and concentrates in metric tonnes

1981	1982	1983	1984	1985	1986	1987	1988	1989	1990	1991	Country/continent
76	53	-	-	-	-	-	-	-	-	-	Germany, FR
252	159	-	-	-	-	-	-	-	-	-	Italy
1560	1540	1416	1520	1539	1471	1533	1499	967	962	52	Spain
67	71	65	80	130	147	144	135	160	140	74	Finland
-	-	52	72	88	75	67	70	51	37	35[a]	Yugoslavia
1955	1823	1533	1672	1757	1693	1764	1704	1178	1139	161	Europe[a]
204	246	162	182	226	262	211	97	202	47	25	Turkey
204	246	162	182	226	262	211	97	202	47	25	Asia[a]
877	386	828	586	801	764	756	662	587	637	431	Algeria
877	386	828	586	801	764	756	662	587	637	431	Africa
3	2	4	2	1	-	-	-	-	-	-	Dominican Republic
240	295	221	384	394	185	124	345	651	735	720[b]	Mexico
962	888	864	657	570	510[b]	140[b]	520[b]	470[b]	500[b]	40[b]	United States
1205	1185	1089	1043	965	695	264	865	1121	1235	760	America
4241	3640	3612	3483	3749	3414	2995	3328	3088	3058	1377	Western countries
153	151	144	152	158	168	164	168	131	126	75	Czechoslovakia
1700	1700	1700	1600	1600	1500	1650	1650	1500	1400	1200	USSR[b]
800	800	850	800	800	850	900	900	1200	930	1000	China, PR[b]
2653	2651	2694	2552	2558	2518	2714	2718	2831	2456	2275	Eastern countries
6894	6291	6306	6035	6307	5932	5709	6046	5919	5514	3652	Total World

Source: Metallgesellschaft 1992; c.f. OECD 1994

[a] Excluding eastern countries (Czechoslovakia, USSR, and China)

[b] Estimated

Table 3. **Metric tonnes of mercury used per year in various categories in Canada and the United States (OECD 1994).**

Use Category	Canada	U.S.A.
Year	1982/91	1989
Batteries	4.3	250.0
Thermometers	4.0	NA
Tooth fillings	10.0	39.0
Pesticides as seed dressing	2.0	NA
Laboratory	4.0	18.0
Hospital equipment	24.0	NA
Measurement equipment	NA	87.0
Electrical:		
Switches	NA	141.0
Light sources	4.5	31.0
Paint	2.0	192.0
Industry:		
chlor-alkali	1.9	381.0
other	NA	40.0
Other uses	NA	35.0
Total use	56.7	1214

NA= not applicable

4.0 SOURCES AND PATHWAYS INTO THE ENVIRONMENT

There are many sources of mercury to the environment, both natural and human related. Natural sources include geological mercury deposits, forest fires and other wood burning activities, volcanoes, and volatilisation from the oceans. The primary human-related sources include: coal and other fossil fuel combustion, chlorine alkali processing, mercury waste in landfills or storage, waste incineration, cement manufacturing and metal smelting. Current estimates suggest that human activities have at least doubled or tripled the amount of mercury in the atmosphere since pre-industrialised times (Krabbenhoft and Rickert 1995). These authors also reported that the atmospheric burden (from all sources) continues to increase by about 1.5% per year. A number of studies have estimated the relative contributions of natural and anthropogenic mercury fluxes to the global atmosphere (Table 4; OECD 1994):

Table 4. Estimates of Fluxes of Mercury to the Global Atmosphere

Process	**Metric tonnes of mercury per year**			
Anthropogenic emissions	2000-10,000	3560 (910-6200)	2000	4500 (3000-6000)
Natural emissions	<15000	2500 (100-4900)	3000-4000	3000 (2000-9000)
Total present emissions	2000-<25,000	6060 (1010-11,100)	5000-6000	7500 (5000-15,000)
Reference	Lindqvist et al. 1984	Nriagu and Pacyna 1988, Nriagu 1989[a]	Fitzgerald 1986	Lindqvist et al. 1991

Notes:

[a] Specific sources are listed in Table 5.

4.1 Natural Sources and Pathways

Various amounts of the mercury are found naturally in rocks and surface materials in all parts of the world. Weathering, leaching and runoff of mercury from soils directly contribute to the mercury in receiving waters. The processes are augmented by deforestation and other forms of land disturbance that promote the release of mercury (Veiga et al. 1994).

The most abundant mercury-containing ores are cinnabar (red HgS), metacinnabar (black HgS) and livingstonite ($HgSb_4S_7$) (D'Itri 1972). The mercury content of the vermilion-red sulphide mineral, cinnabar exceeds 86%. It occurs in high quantities in relatively few locations in the world (Mitra 1986), although not all countries that have large deposits of mercury are involved in its mining. Rocks and soils commonly have concentrations of between 5 and 1000 $\mu g \cdot kg^{-1}$ with highest levels near cities and some of the lowest levels in cultivated chernozemic soils of the Canadian prairie provinces (Anderson 1979).

Major mercuric sulphide deposits in Canada are located mainly in western Canada (with one occurrence at Clyde Forks, Ontario). In British Columbia significant economic grade deposits of mercury are known (Nagpal 1989), with concentrations of more than 1 $g \cdot kg^{-1}$ in the Kamloops, Port Clements, Bridge River and Yalakom River areas (AEP 1992). No mercury mines are currently operating in Canada (A. Plouffe 1999, GSC, pers. com.).

The volatile nature of mercury makes degassing of the earth's mantle and volcanic emissions important natural sources of mercury to the atmosphere (U.S. NAS 1977). Oceanic submarine volcanoes (Varekamp and Buseck 1986), and seismic activities like earthquakes (Varshal et al. 1985) are other natural sources of mercury. Biological formation of volatile elemental mercury from methylmercury is also recognised as a source of Hg to the atmospheric pool (Lindqvist et al. 1991).

While the vast majority of natural mercury emissions is from the natural weathering of rocks and mineral deposits and degassing, mercury is also released to the atmosphere through forest fires and other wood burning activities. For example, annual releases to the atmosphere from natural fires in the boreal forest region of Manitoba was calculated to be 20 g Hg·ha^{-1} representing 0.02% of the provincial annual emissions from natural sources which creates high short-term emissions in the form of a pulse. The evaluation assumed 0.4 ppm as the Hg concentration in timber, but about 0.08 ppm was considered transferred to the atmosphere during fires (Williamson 1986). Worldwide, forest fires are estimated to be responsible for releasing 20 t of Hg to the atmosphere each year, which is less than 1% of one researcher's estimate of natural emissions (Nriagu 1989). Intentional wood combustion was estimated to represent 60 to 300 t of Hg (about 5% of all anthropogenic emissions in 1983).

Different types of lakes are associated with proportionally different species and sources of mercury and methylmercury (MeHg). In seepage lakes (no surface inflow), essentially all MeHg is formed within the lake (Watras et al. 1994). In drainage lakes, significant inorganic mercury or organic mercury may be transported from the drainage basin to the lake complexed with dissolved organic carbon (DOC) or particulate matter (Lee and Hultberg 1990; Lee and Iverfeldt 1991), thereby supplementing direct atmospheric sources and in-lake net methylation. The processes of MeHg production and transport to aquatic systems will be discussed more fully in chapter 5.

Watersheds tend to be net sinks of mercury, with the amount of mercury retained often depending on the proportion of wetlands in the watershed (Mierle 1995). More mercury and methylmercury are exported downstream from watersheds dominated by wetlands because of complexation with dissolved organic matter (Mierle and Ingram 1991; Engstrom et al. 1994; Rudd 1995). Also, more mercury usually enters lakes that have a large drainage basin relative to the lake area. The same relationship to high $A_{dr.}/A_{lake}$ has been associated with increasing mercury levels in some Ontario fish (Suns and Hitchin 1990).

4.2 Anthropogenic Sources

Of the significant sources of mercury to the Canadian environment; smelting, municipal solid waste and sewage sludge incineration, fossil-fuel burning, and cement manufacturing are presently considered the most important.

From all Canadian sources, mercury emissions were estimated at 40 t during 1978, 31 t in 1982 (Jaques 1987), 33 t (Environment Canada 1997; L. Trip 1999, Environment Canada pers. com.), or 43 t (EPRI 1996) in 1990, and declined to 12 t in 1995 (Environment Canada 1997; L. Trip). The decline by 1995 was principally due to the closure of a smelter in Flin Flon, Manitoba that resulted in reducing overall smelter emissions to about 26% (<5 t) of total mercury emissions in Canada. It also reduced the estimated overall national emissions by more than 50%. Emissions of mercury to the atmosphere as of 1995 totalled approximately 143 t for the U.S. (Pilgrim and EMAN 1998), down from 280 t in 1990. In 1990, about two-thirds of Canadian emissions (22 t) were from non-ferrous metal smelting. The next highest emission sector in 1990 was the incineration of municipal solid waste and sewage sludge, at 9.4% (of 33 t), or 3.1 t (L. Trip, unpubl. data). Anthropogenic mercury emissions in Canada for all sectors in 1990 compared to 1995 are shown in Figure 1, and emissions for each province in 1990 are shown in Figure 2[4].

Emissions reported to the National Pollutant Release Inventory (NPRI) in 1995 totalled 2.4 t of mercury (NPRI 1997). The highest reporting emitter was Cominco Ltd. in Trail, BC, which released 1.8 t to the air, and 60 kg to the Columbia River. All other industries in Canada combined, reported a total of 476 kg to air and 11 kg to water (NPRI 1997). Reported emissions are only a fraction of the total releases of mercury to the environment that actually occur. The discrepancy is mainly due to other sources including fossil fuel combustion that are not covered under the NPRI[5], but which are included in the Canada-wide estimates discussed above (L. Trip 1999, Environment Canada, pers. com.).

On a global scale, the major air emissions of mercury originate from coal combustion and incineration of solid waste products, whereas the major emission to water is from manufacturing processes. Nriagu and Pacyna (1988) and Nriagu (1989) estimated the worldwide emissions of mercury to the atmosphere, soil and water for 1983, listing all anthropogenic plus total natural sources (Table 5). Even though the anthropogenic component has decreased as use has declined, some researchers agree that anthropogenic sources still comprise about half of the global emissions (see Flux estimate table above; Lindqvist et al. 1984, 1991; Fitzgerald 1986).

Of atmospheric mercury originating from anthropogenic sources, worldwide fossil fuel combustion is estimated to provide one-half, while the rest is released through volatilisation of fungicides, paints, manufacturing, mining, waste incineration, battery decay, fluorescent lamps and various other sources (Douglas 1991). Fewer chlor-alkali operations exist than in former times, but coal-fired power plants and petroleum product combustion (gasoline, oil, etc.) that release mercury in the vapour phase directly to the atmosphere, continue to be important sources. An estimate reported annual global emissions at 2214 t as of 1990. That estimate reported that Asian countries combined emit the highest tonnage of mercury to the atmosphere, at 44% of global emissions (976 T; L. Trip 1999, Environment Canada pers. com.). As of 1990, North America, western Europe, and eastern Europe plus the former USSR countries each emitted about 15%, or 330-349 t of mercury (Figure 3). Of the 1990 total, North American emissions of 330 t, the United States emitted 84.9%, Canada emitted 9.4%, and Mexico emitted 5.7% (Figure 3).

Mercury in solid waste is a potentially preventable source of environmental mercury contamination. For example, the overall quantity of mercury in the U.S. in municipal solid wastes was predicted by US EPA (1992) to decrease through the 1990's, falling from a total of 709 tons in 1989 to 173 tons in the year 2000 (Table 6)[6]. The amount of mercury contributed by batteries was also predicted to decrease, from approximately 621 tons in 1989 to 98 tons in the year 2000, largely due to reduced use of mercury in these products. In contrast, US EPA projected that mercury in solid waste from electrical lighting would slowly increase, rising from approximately 27 tons in 1989 to 41 tons in the year 2000 (Smith and Rowan-West 1996).

Mercury in solid waste in Canada has not been quantified (G. Ironside, Environment Canada, pers. com.). Assuming that Canadian usage of mercury is similar to the U.S, the values cited for the U.S. in the previous paragraph may be reduced by a factor of about ten to estimate amounts of mercury in solid waste in Canada.

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[4] 1995 provincial emission data were unavailable as of the time of writing (L. Trip).

[5] Appendix containing detailed a) national compilation of discharge and emission data; and b) control/treatment technology data and information is in preparation.

[6] Mercury mining operations in the U.S. ceased in 1991. In 1994, the U.S. suspended sales of its stockpile mercury.

Even though many point sources of mercury have been removed, long-range transport of mercury in the atmosphere remains elevated in North America in comparison with pre-industrial times (Engstrom et al. 1994). One global estimate suggested that about one-third of atmospheric mercury came from the oceans, and one-third resulted from natural terrestrial processes like degassing and geologic weathering (Fitzgerald 1986). The remaining atmospheric sources were estimated to be from the production, use and disposal of mercury by humans.

Mercury releases and usage are declining in Canada and the United States, but because of the persistence of mercury compounds in the environment, former discharges contribute to the current atmospheric loading and redistribution of mercury in the environment. Mercury sources, uses and recycling in the environment are reviewed in Nriagu (1979, 1989), IJC (1990), AEP (1992), Watras et al. (1994), and Rudd (1995).

At a recent meeting of the Scientific Committee on Problems of the Environment (SCOPE, Stockholm 1993), it was concluded that anthropogenic sources account for approximately 50% of atmospheric mercury (Jernelöv and Ramel 1994). The Expert Panel on Mercury Atmospheric Processes (EPMAP), which convened in March 1994, suggested that as much as 75% of total emissions were from anthropogenic sources (EPMAP 1994). EPMAP also reported that historical deposition in lake and peat sediments indicate that global atmospheric concentrations of mercury have increased by a factor of about three (rather than a factor of two as discussed above) since pre-industrial times. All experts agree that precise estimates are difficult to make because it is currently impossible to accurately assess the recycled portion of mercury compounds emitted from the earth's surface including the oceans.

Table 5. Estimates of worldwide emissions of mercury (metric tonnes per year) to the atmosphere, soil, and water in 1983 (from Nriagu and Pacyna 1988 and Nriagu 1989).

Source Category	Atmosphere[a]		Water		Soil[b]	
	min.	max.	min.	max.	min.	max.
Agricultural waste	no estimate		no estimate		0	1700
Atmospheric fall-out	no relevance		220	1800	630	4300
Coal combustion	650	3500	0	3600	370	4800
Dumping of sewage sludge	no relevance		10	310	no relevance	
Logging and other wood wastes	no estimate		no estimate		0	2200
Manufacturing Processes	no estimate		20	2300	no estimate	
Metal mining	Insignificant input		0	150	no estimate	
Mine tailings	no estimate		no estimate		550	2800
Non-ferrous metal production	45	220	0	40	0	80
Phosphate fertilizer production and use	Insignificant input		no estimate		no estimate	
Refuse incineration			no estimate		no estimate	
Municipal	140	2100				
Sewage sludge	15	60				
Smelter slags and wastes	no estimate		no estimate		50	280
Urban refuse	no estimate		no estimate		0	260
Wastage commercial products	no estimate		no estimate		550	820
Wastewater	no relevance		0	600	10	800
Wood combustion	60	300	no estimate		no estimate	
Total inputs (Median)	910	6180 (3560)	250	8800 (4600)	2140 (8300)	18040
Natural (Median)	100	4900 2500	no estimate		no estimate	

Notes:

[a] Insignificant contributions to the atmosphere from: oil combustion, zinc-cadmium production, secondary non-ferrous production, steel and iron manufacturing, cement production, and mobile sources.

[b] Landfills included

Table 6. **U.S. nation-wide discards[a] (tonnes per year) of mercury in products in municipal solid wastes (MSW), 1970 to 2000**

Products	1970	1980	1989	1995	2000
Dental Uses	9.3	7.1	4.0	2.9	2.3
Electric lighting	19.1	24.3	26.7	33.6	40.9
Fever Thermometers	12.2	25.7	16.3	16.9	16.8
Film Pack Batteries	2.1	2.6	0.0	0.0	0.0
Household batteries	310.8	429.5	621.2	176.6	98.5
Mercury Light Switches	0.4	0.4	0.4	1.9	1.9
Paint residues	30.2	26.7	18.2	2.3	0.5
Pigments	32.3	23.0	10.0	3.0	1.5
Special Paper Coating	0.1	1.2	1.0	0.0	0.0
Thermostats	5.3	7.0	11.2	8.1	10.3
Total discards	421.8	547.5	709.0	245.3	172.7

Sources: US EPA 1992; Smith and Rowan-West 1996.

[a] Discards before recovery (some MSW may be recovered for recycling).

5.0 ENVIRONMENTAL FATE AND BEHAVIOUR

While chapter 4 described the sources of mercury and the "big picture" of its transport in the environment, this chapter will review the smaller scale processes involved in the behaviour of the various mercury species. In ecosystems, the form of mercury and its mobility is dependent on environmental characteristics. Site-specific factors that control the net production (balance of methylation and demethylation), photodegradation, and distribution of MeHg between gaseous, dissolved and solid phases are of particular importance because MeHg is much more toxic and more readily bioaccumulated than any other form of mercury.

The mercury cycle describes the fate, behaviour, and transport of mercury in the environment. For the purposes of this report, fate is defined as the quantification and movement of various mercury species among environmental phases (i.e., gaseous, water, colloids, suspended particulates, sediment, or biota) in lakes and watersheds. Sorption, desorption, diffusion, volatilisation and transformation (reduction, methylation, demethylation) are among the processes that the various mercury species undergo.

For inorganic mercury, the most toxicologically relevant process is methylation. For MeHg, processes that define its fate as a species include demethylation and photodegradation. Diffusion of MeHg through biological surfaces and bioaccumulation are components of the mercury cycle that will be discussed more fully in the following chapter.

5.1 The Mercury Cycle

Chemical cycling is defined as inputs to and outputs from a system, along with transport and transformation of chemical species within the system (Zillioux et al. 1993). Transport of mercury, and especially MeHg, among various abiotic and biotic compartments is a concern when it threatens the health of aquatic biota or humans.

The mercury cycle in aquatic ecosystems is complicated by numerous possible mercury species that are present, and by variables affecting speciation. In the last decade, there was a marked increase in our knowledge of environmental concentrations of mercury species because of the development of strict clean protocols for the sampling and improvements to the analysis of mercury and MeHg (Gill and Fitzgerald 1985;1987; Driscoll et al. 1994). Typical total mercury concentrations in 'uncontaminated' lake water (epilimnetic) in the northern U.S. are about 0.5-4 $ng{\cdot}L^{-1}$ (Hurley et al. 1994; Watras et al. 1994), with MeHg making up a small proportion of the total.

Generally, the aquatic mercury cycle may be characterised by high atmospheric fallout, removal by deposition to sediments, re-emission to the atmosphere as elemental Hg (Hg^0), methylation and demethylation transformation processes within the lake or watershed (Figure 4; Watras et al. 1994 1995) and exchanges through tributaries and groundwater (runoff and seepage; Hurley et al. 1994). Some portion of the total mercury pool is accumulated by biota, with biomagnification of MeHg in higher trophic levels of food webs.

Atmospheric fallout directly provides most of the mercury to watersheds that are uninfluenced by point sources or high natural source contamination (Figure 4). Wet and dry fallout of mercury to lakes occurs largely as inorganic mercury (mainly Hg^{2+}; Winfrey and Rudd 1990), with insignificant inputs of MeHg (Bloom and Watras 1989). Long-range atmospheric transport of mercury results in mercury deposition to environments thousands of kilometres from emission sources, although about half of the fallout originates within 1000 km (EPMAP 1994).

In surface waters, mercuric ions (Hg^{2+}) may be reduced microbially to form elemental mercury (Ramamoorthy et al. 1983). Photochemical reduction may also occur with exposure to UV_B in sunlight (Amyot et al. 1994). Most surface waters are oversaturated with Hg^{0} and because it is volatile, it is likely to be re-emitted to the atmosphere (Vandal et al. 1991; Driscoll et al. 1994). Small amounts of MeHg may also volatilise (especially dimethylmercury), but Hg^{0} is by far the largest component of the total gaseous mercury concentration in air (Schroeder et al. 1991). Re-emissions are important sources of mercury to the global cycle and can result in oxidation to Hg^{2+} and re-entry to surface waters in association with precipitation or dry fallout (Figure 4).

Oceanic production and subsequent emission of Hg^{0} is a particularly important facet of the global Hg cycle. This is particularly true near ocean margins (Cossa et al. 1996). The oceans receive about 90% of their mercury as Hg^{2+} from wet and dry atmospheric deposition. Elemental mercury forms about 5 to 30% of the total Hg in ocean waters (Mason et al. 1994). Biological production of Hg^{0} has been reported in the open ocean, coastal mesocosms and in permanently anoxic fjords in Norway (Vandal et al. 1991). While re-emissions of the volatile Hg^{0} result in a loss of reactive Hg species (Hg^{2+}) from a location where they might become methylated, the continual recycling of Hg back to the atmosphere prolongs the impact of anthropogenic Hg on aquatic systems. This is because while most of the Hg deposited to the ocean is recycled to the atmosphere, the terrestrial environment and freshwater ecosystems become the principal atmospheric Hg sinks (Mason et al. 1994).

Once in lakes, most mercury binds to particulates and is quickly deposited to sediments, where it may be buried or transformed. Of all of the binding sites, particulates are considered to be most significant and may result in the efficient removal of mercury from the water column by sedimentation. Methylmercury, and especially $HgCl_2$, bind quite strongly with suspended particulates (Table 1). After deposition, the mercury is found complexed with the sulphide as HgS, or adsorbed on organics, clays, and Fe or Mn oxides (Miller 1975; Jackson 1989).

The deeper water near the sediment-water interface in lakes is often enriched in mercury compared with other zones in the lake. Because of the finding that particulate-associated mercury concentrations were higher in the deeper layers than the shallow layers of stratified lakes, it was concluded that allochthonous (source outside the lake) Hg was scavenged by particles in the upper waters and transported down with settling particulates (Watras et al. 1994). The alternative was that deeper waters were enriched in particle-associated mercury because of resuspension from bottom sediments. Resuspension can occur due to turbulence from water currents, or bioturbation. In sediments, tubificids, clams, chironomids, other invertebrates, and even fish have been known to reintroduce mercury to the water column by disturbing the sediments (Andersson et al. 1990).

Porewater diffusion (movement through sediments) of mercury is undocumented for many types of lakes. Measurements of sediment porewater in a seepage lake indicated that diffusional movement of total mercury averaged $1.4 \cdot 10^{-8}$ $ng \cdot cm^{-2} \cdot s^{-1}$ (concentration gradient of 15-35 $ng \cdot L^{-1}$; Hurley et al. 1994). The authors considered that the diffusional flux from sediments to the water column was minor compared to redissolution of recently fallen particulate material at the sediment surface. Compared with other metals like iron, mercury release from bottom sediments is not nearly as highly redox-dependent (Hurley et al. 1991).

Runoff and seepage of mercury associated with particulates or dissolved organic matter from watersheds may also introduce both inorganic and methylated forms of mercury to lakes and rivers. Substantial increases in total mercury and MeHg may occur when groundwater discharges through wetlands into streams or from rivulets that drain across the surface of wetlands (Krabbenhoft et al. 1995). Once in surface waters, mercury may enter sediments as described above, be transformed (e.g., methylated or demethylated) and/or be transferred to biota. More so than inorganic mercury, MeHg has some affinity for organic matter and biological tissue[7] because of its attraction to sulphydryl groups in proteins, thus may become preferentially concentrated in fish and other aquatic biota.

Numerous studies have indicated the importance of organic carbon in binding and transporting mercury compounds (Ramamoorthy and Kushner 1975; Lee and Iverfeldt 1991; Mierle and Ingram 1991; Miskimmin 1991; St. Louis et al. 1994). There is little question that dissolved organic carbon (DOC - often measured as "colour") can increase the apparent solubility of mercury. For example, the export of mercury from watersheds to lakes is closely tied to the export of humic material (DOC; Mierle and Ingram 1991). Hurley et al. (1995) found a positive correlation between the percent wetland in the watershed and MeHg yield to rivers. In northern Ontario, even though upland watersheds without wetlands yielded more total mercury per area to lakes, high DOC wetland portions of watersheds yielded 26-79 times more MeHg than uplands (St. Louis et al. 1994). Since the proportion of MeHg that is transported from watersheds is dependent on biotic and abiotic MeHg production, abundant mercury methylation probably occurs in wetland environments, where high organic carbon and bacterial activity are typical.

Seasonally, mercury in the water column is lower in winter than in summer because frozen lakes are sealed off from atmospheric sources of mercury and most particulate settling has occurred (Watras et al. 1994). There is also a seasonal component to methylmercury cycling. Methylmercury concentrations in water were found to be higher in the late summer than at other times of the year in both rivers (Hurley et al. 1995) and lakes (Watras et al. 1995). Ramlal et al. (1993) observed the same seasonal trend for measured net methylation rates in sediment cores from northern Ontario. Methylmercury concentrations were highest by late in the summer because of the high bacterial activity associated with higher temperatures as the summer season progressed, causing MeHg to accumulate. Methylation but not demethylation was stimulated by warm summer temperatures; while the opposite was true in cold winter conditions (Ramlal et al. 1993). In general, net MeHg production in lakes is increased by factors that enhance bacterial activity, and total Hg is reduced when lakes are sealed off from atmospheric sources. Reviews of the aquatic mercury cycle may be found in Driscoll et al. (1994) and Zillioux et al. (1993). Also, a mathematical model of mercury cycling in lakes was developed by Hudson et al. (1994).

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[7] MeHg has relatively low log Kow values of 1.6 ± 0.2 (Major et al. 1991) and 2.5 (Halbach 1985).

5.2 Mercury Methylation and Demethylation

Before the mid-1960s, inorganic mercury was believed to be relatively inert in the environment. Then it was demonstrated that inorganic mercury could be methylated by micro-organisms to form MeHg, which was biomagnified in food webs (Wood et al. 1968; Jensen and Jernelöv 1969). The production of methylmercury is a balance between methylation and demethylation. Wherever MeHg is detected in the environment, methylation has exceeded demethylation.

Since methylation and demethylation always co-occur, the balance between the two in the aquatic ecosystem is called net MeHg production. Several factors influence the rate of net MeHg production in water. These include the concentration of Hg^{2+}, composition of the microbial population, nutrient and mineral substrate, pH, temperature, redox potential, dissolved and particulate organic matter, iron, and sulphate. Methylation by naturally-occurring micro-organisms is probably limited by processes that alter the availability of inorganic mercury (Ramamoorthy et al. 1982; Winfrey and Rudd 1990). Early studies reported that micro-organisms produced MeHg under anaerobic conditions (Jensen and Jernelöv 1969). Subsequently, aerobic methylation was demonstrated (Rissanen et al. 1970), and it is now widely accepted that methylation occurs in both aerobic and anaerobic environments.

MeHg is formed principally through the microbial transfer of methyl groups (CH_3) to inorganic mercury (e.g., Hg^{2+} transformed to CH_3Hg^+; Robinson and Tuovinen 1984). Methylcobalamin is thought to be the only non-enzymatic methylation agent capable of transferring a methyl group to an inorganic mercuric ion. It can transfer groups as a carbanion (CH_3^-) and a methylradical ($CH_3\bullet$) to produce methylmercury and dimethylmercury (D'Itri 1991), as follows:

$$Hg^{2+} \xrightarrow{CH_3B_{12}} \underset{\textit{Mono-MeHg}}{CH_3Hg^+} \xrightarrow{CH_3B_{12}} \underset{\textit{Di-MeHg}}{(CH_3)_2Hg}$$

Under anaerobic conditions, methylcobalamin was shown to be present and the reaction to require ATP and hydrogen as the source of electrons (Wood et al. 1968; Robinson and Tuovinen 1984). Three major co-enzymes may participate in enzymatic methylation, but the process remains unvalidated (D'Itri 1991).

While biotic methylation is the dominant process, some abiotic mercury methylation also occurs with methyl derivatives of lead, tin and silicon (AEP 1992), and in the presence of high concentrations of humic matter ($6380\ mg \cdot L^{-1}$; Nagase et al. 1982) along with metal ions (humics $>170\ mg \cdot L^{-1}$; Lee et al. 1985).

Demethylation was first discovered by Furukawa et al. (1969), at about the same time that methylation was reported. Demethylation is the degradation of MeHg involving the microbial cleavage of the carbon-mercury linkage followed by the reduction of Hg^{2+} to Hg^{0} (Robinson and Tuovinen 1984). In addition to metallic mercury, methane is a by-product of demethylation. The biochemistry and genetics of the plasmids and associated enzymes involved in mercury demethylation were thoroughly reviewed by Summers and Silver (1978); Robinson and Tuovinen (1984); Summers (1986); and Foster (1987).

While non-biological demethylation mechanisms are unknown (Winfrey and Rudd 1990), a newly discovered process is the breakdown of MeHg by photodegradation (Sellers et al. 1996).

Microbial demethylation has long been accepted as the dominant route of methylmercury degradation in fresh waters. It is not known whether photodegradation results in the production of the volatile Hg^0. Under the conditions of the experiments of Sellers et al. (1996), however, photodegradation of MeHg was about 350 times greater than microbial demethylation. These results suggest that MeHg degradation may be an important process where light penetration is significant (such as in clear and/or shallow water).

A number of environmental variables affect the net production of methylmercury. Most factors that increase rates of bacterial activity will enhance MeHg production. Bacterial activity increases with increasing temperature and available biodegradable organic carbon. Thus, methylation rates tend to be highest in surface sediments with freshly deposited organic matter, and in warm shallow sediments where abundant bacterial activity takes place (Ramlal et al. 1986; Winfrey and Rudd 1990). Methylation also occurs in the aerobic water column (Furutani and Rudd 1980; Xun et al. 1987), in anaerobic water and sediments (Ramlal et al. 1985), fish intestinal contents and surface biofilms (*in vitro* studies; Rudd et al. 1980).

Some lakes are more prone to MeHg problems than others. These include hydroelectric reservoirs (Abernathy and Cumbie 1977; Lodenius et al. 1983; Stokes and Wren 1987; Jackson 1988a; Hecky et al. 1991), low pH lakes (Wren and MacCrimmon 1983; Verta et al. 1986; McMurtry et al. 1989), and even some remote lakes with no apparent other stressors associated with high mercury. The explanation for high MeHg in fish from these lakes is different in each case, but usually relates to factors affecting net MeHg production and transfer to the food web. In the case of newly formed reservoirs, the flooding of fresh vegetation and subsequent high bacterial activity causes mercury methylation to increase substantially (Ramlal et al. 1987; Hecky et al. 1991). The elevated MeHg in fish may decline in a few years or may remain elevated for decades (Bodaly et al. 1984). Prediction of mercury problems associated with reservoirs may be possible using mathematical modelling (Johnston et al. 1991).

In low pH and other remote lakes, the explanation for high MeHg in fish is more complex. Mercury methylation rates are higher in low pH lake water and surface sediments (Xun et al. 1987). In fact, the methylcobalamin-dependent methylation process is optimised at pH 4.7 (D'Itri 1991). It is also possible that the uptake of MeHg by aquatic organisms in acidic waters is increased by calcium mediated changes in gill permeability (Rodgers and Beamish 1983). Further, the importance of sulphate reducing bacteria is enhanced in culturally acidified lakes; these bacteria are thought to be the most important methylators in lake sediments (Gilmour and Riedel 1995). In remote non-acidified lakes with no apparent point source of mercury, sometimes the 'source' is traced to wetlands which may have high methylation rates combined with high DOC-related export of MeHg, as discussed in section 5.1 (The Mercury Cycle).

5.3 Factors Affecting Partitioning and Speciation

The partitioning of mercury between "dissolved" and "particulate" phases is directly related to aqueous speciation and to lake chemistry. Partitioning of a given species is the outcome of the competition for that species among ligands in solution (truly dissolved), small particles called colloids, and organic or inorganic material. Speciation describes whether the form of mercury is an alkylated form, or is one of the many inorganic forms (e.g., $HgCl_2$, HgS, etc.). This document will only discuss MeHg and a few of the important inorganic forms. Full reviews and examples of mercury speciation research may be found in Gill and Bruland (1990), Aceto et al. (1995), and in several papers within the book, "Mercury Pollution: Integration and Synthesis" (Watras and Huckabee 1994).

The relative abundance of MeHg in environmental samples is of particular concern because of its high toxicity to biota. Methylmercury is the major form of mercury accumulated in biological tissues due to the ease with which it permeates cell membranes and its strong affinity for sulphydryl groups in proteins (Faust 1992). It is not highly lipophilic, with moderate K_{ow} values of up to 2.5 found experimentally (Table 1). In water samples, MeHg is typically a smaller proportion of total mercury than is inorganic mercury. In low alkalinity lakes, MeHg has been reported to represent 10-20% (Adirondacks, Driscoll et al. 1994), and <1-13.3% (lake inflows, Precambrian Shield of Ontario; St. Louis et al. 1994) of total mercury in water samples. An unusual case was for moderately alkaline (up to 3 $meq \cdot L^{-1}$ or 150 $mg \cdot L^{-1}$ $CaCO_3$), high pH (8-9) lakes in California, where up to 89% of total Hg was MeHg (Gill and Bruland 1990).

The effect of pH on mercury speciation relates partly to its effect on the solubility of inorganic mercury and partly to the finding of higher rates of MeHg production with decreasing pH (Xun et al. 1987; Miskimmin et al. 1992). With decreasing pH, all forms of mercury show increased binding to sediments (where bacteria are abundant). A different relationship between pH and DOC may be important in regulating the availability of inorganic mercury for methylation. Reduction in pH changes the character of DOC by increasing protonation of anions and thus desorbing metals (Davis et al. 1985). If binding of inorganic mercury to some forms of DOC is reduced by low pH, more mercury may be available to micro-organisms for methylation (Miskimmin et al. 1992).

Redox conditions and chloride ions influence Hg speciation and methylation (Gill and Bruland 1990). In well-aerated water (redox ≥0.5V), mercuric (+2) species will predominate, whereas under reducing conditions, elemental (Hg^o) species will occur (AEP 1992). Abundant MeHg may be produced by anaerobic (e.g., Ramlal et al. 1985), as well as aerobic bacteria. Among the inorganic anions, Hg^{2+} forms the strongest covalent bond with chloride ions (stability constant, $K=10^{15}$), so mercuric-chloride species may dominate when chloride salts are abundant (AEP 1992). Chloride salts may be locally important where runoff from road salting operations occur.

Within anoxic zones, inorganic mercury forms strong aqueous complexes with sulphide and precipitates as HgS (Craig and Bartlett 1978; Leermakers et al. 1993). These sulphide complexes are generally assumed to control speciation of both Hg^{2+} and MeHg. While sulphate reducing bacteria are known to methylate mercury (Gilmour et al. 1992), sulphate reducers make up only a small proportion of the microbial community in most freshwater lakes, where methanogens are the primary anaerobic bacteria. Sulphide is considered important because it out competes humic acid (DOC) by factors of 10^4 for CH_3Hg^{2+} and 10^{18} for Hg^{2+} (Hudson et al. 1994). Hurley et al. (1994) completed extensive calculations of the equilibrium speciation of mercury and sulphur at a range of redox and pH levels.

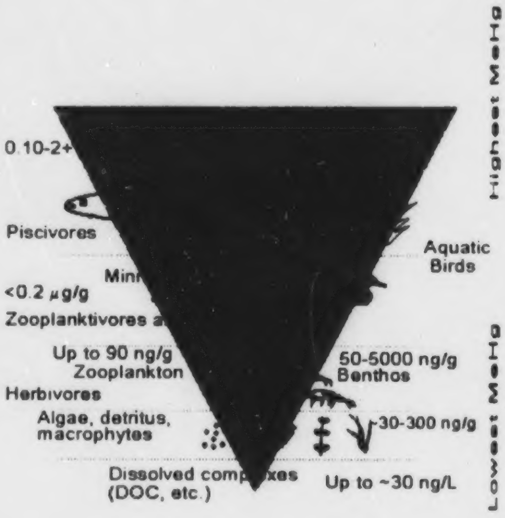
Potential sulphide binding that reduces bioavailable mercury may be particularly important in the saline lakes (>3 $g \cdot L^{-1}$ salt) of Alberta and Saskatchewan which have some of the highest sodium sulphate concentrations in the world (Na_2SO_4 >125,000 µM; Waiser and Robarts 1995). HgS precipitation combined with high pH (associated with low methylation rates), decreased methylation with increasing salinity (Blum and Bartha 1980) and potential Hg^o re-emissions (high from shallow, wind-mixed waters) makes significant MeHg bioaccumulation an unlikely problem in prairie saline lakes. Perhaps an analogous situation is in the high pH, high-alkalinity desert lakes in California, where very little or no MeHg was detected using highly sensitive analytical methods (0.5 $pg \cdot L^{-1}$ detection limit; Gill and Bruland 1990).

6.0 BIOACCUMULATION OF MERCURY AND METHYLMERCURY IN AQUATIC AND SEMI-AQUATIC ORGANISMS

Methylmercury has a high potential for bioaccumulation as well as biomagnification with increasing trophic levels. Mercury and MeHg may be taken up by aquatic organisms directly from water or through their diet. Uptake directly from water is the result of dissolved mercury adsorption or absorption through the body surface and respiratory organs such as gills. Uptake via food is based on mercury transfers through the digestive tract. The relative importance of either pathway depends on trophic level of the organisms, duration and intensity of exposure, and environmental factors (Zillioux et al. 1993).

Organisms at lower trophic levels usually contain the lowest proportion of total mercury as MeHg. Aquatic plants contain the lowest MeHg as a percentage of total mercury. Invertebrates often contain about 50% MeHg and 50% inorganic mercury (Hildebrand et al. 1980). Organisms higher in the food chain, like piscivorous fish (e.g., walleye, lake trout), aquatic birds (loons, herons), piscivorous mammals (mink, otters) and marine mammals contain a high proportion of THg as MeHg in muscle tissue. Most piscivorous fish have essentially all MeHg in their muscle tissue (Bloom 1989). The main route of uptake for MeHg is consistent with a number of persistent organic chemicals like DDT, PCBs or dioxins. Once debated, it is now generally accepted that uptake through diet sources is the most important route of uptake for fish (Rodgers 1994) and, for wildlife consumers of fish. Methylmercury accumulates in organic tissue because of its affinity with sulphydryl groups, the relative ease with which it passes through the digestive wall and slower depuration rate relative to inorganic mercury (Saouter et al. 1993).

MeHg Biomagnification in the Food Web



6.1 Aquatic Plants

Direct adsorption of mercury is the critical pathway for phototrophic organisms like macrophytes and algae. Small algal cells may accumulate more mercury than large ones because of the higher surface area to volume ratio for small cells compared to large cells. Methylmercury is preferentially sequestered in algal cytoplasm while inorganic Hg is retained in the algal membrane (Mason et al. 1995). This has implications for the transfer of MeHg to zooplankton, to be discussed in the next section (6.2).

Because phytoplankton cells continually divide, the partitioning of Hg into live cells is a balance between uptake, depuration and growth dilution. This is fundamentally different from adsorption of Hg onto detrital or inorganic particles. Plankton may potentially accumulate more Hg than does detritus because the living cells have both passive and facilitated transport mechanisms, as well as intracellular ligands that sequester Hg (Hudson et al. 1994).

The reported data on mercury uptake by aquatic macrophytes are sometimes contradictory, with some studies indicating that mercury accumulation takes place primarily in roots while others indicating accumulation is primarily through rhizomes or shoots. Given that rooted macrophytes accumulate the majority of their nutrients through their roots and the fact that mercury concentrations are higher in sediments than in water, it may seem intuitive that more mercury would be accumulated through the roots from sediments. Jana (1988) found twice as much mercury in roots compared to shoots of water hyacinth *(Eichhornia crassipes)* and Coquery and Welbourn (1995) found seven times more total mercury in the roots than in the shoots of a submergent perennial in Ontario. On the other hand, Thompson (1996) found five times and three times more total mercury in the petioles and leaves, respectively, than in the roots of a floating leaved macrophyte [the yellow pond lily (*Nuphar variegatum*)], further fuelling the debate over "root versus shoot" absorption. Since the majority of macrophyte growth is in the shoot, mercury may also be taken up into new cells directly from the water column. Mortimer (1985) and Crowder (1991) felt that the confusion over "roots versus shoots" may have come from the fact that mercury may be translocated from one portion of the macrophyte to another. Regardless, the important point is that if significant amounts of mercury are accumulated by plants, the mercury will be released to the environment upon senescence, or transferred to animals that consume them.

With MeHg, while high accumulation was measured in leaves, stems and roots of *Elodea densa*, root absorption was reported to be the dominant direct route of uptake, and the leaves were the principal storage organ (Maury et al. 1988).

Environmental variables can affect the bioaccumulation of mercury by plants. Coquery and Welbourn (1995) found that total mercury in macrophyte roots was related to mercury in sediments, but only after correction for sediment organic content. If organic content of the sediments was high, mercury concentrations of the sediments could also be elevated without significant uptake by the plant. In that study, concentrations of mercury in plants were greater than the concentration in sediments, and mercury in roots was consistently higher than in the shoots of *Eriocaulon septangulare* (0.22 $\mu g \cdot g^{-1}$ and 0.03 $\mu g \cdot g^{-1}$ dw, respectively). Increasing temperature and photoperiod exert a strong positive influence of MeHg accumulation to macrophytes, with the effects amplified when both are increased concurrently (*Elodea densa*; Maury-Brachet et al. 1990). Differences in accumulation of mercury may also depend on the species of plant, type and amount of complexing ligands and seasonal growth rate changes (Czuba and Mortimer 1980).

Experimentally, the presence of DOC (in the form of humic acid or EDTA) significantly suppressed dissolved mercury uptake by duckweed (Mo et al. 1989). However, in a field survey of 34 Precambrian Shield lakes in Ontario and Québec, algal mercury concentrations showed a significant positive correlation with DOC and with fish mercury concentrations even though mercury in the water was at low to undetectable concentrations (Stokes et al. 1985). The difference between experimental and field results is probably because while DOC may suppress uptake, high DOC lakes often contain more mercury from terrestrial/wetland sources, as discussed section 5.1.

Furthermore, the total mercury concentration in fifty-five *Myriophyllum spicatum* plants has been shown to be negatively correlated with the pH of the water (Thompson-Roberts et al. 1999). In the same study, DOC, alkalinity, and pH were not related to the total mercury concentrations in three other aquatic macrophytes. The authors believed the lack of relationships were due to the small range in environmental variables.

Bioaccumulation is proportional to dissolved concentrations assuming that the rates of uptake by plants are similar for inorganic and organic forms (Mortimer 1985). Aquatic plants are known to bioconcentrate mercury and MeHg up to several thousand times the water concentration (per g dw). Laboratory studies on four genera of submergent macrophytes from the Ottawa River had bioconcentration factors (BCFs) for total mercury ranging from 900-3300 ($\mu g \cdot g^{-1}/\mu g \cdot mL^{-1}$; water concentration 0.28 $\mu g \cdot mL^{-1}$, Mortimer 1985). Similarly, in the field, floating macrophytes (*Nuphar variegatum*) from forty-five Ontario wetlands had BCFs for total mercury ranging from 400 to 6000 (Thompson 1996; Thompson-Roberts et al. 1999). Calculations from Jana's (1988) data resulted in BCFs of 670 for an alga, and 800 for water hyacinth.

Mortimer (1985) proposed that concentrations of mercury in plants water could be used to estimate those in water; for example, if an *Elodea* plant contained 300 $ng \cdot g^{-1}$ dw, then the water in which it was growing would contain 20 $ng \cdot L^{-1}$ based on a BCF of 1500. Mortimer (1985) further stated that since approximately 30% of total Hg was MeHg, the water concentration of both MeHg and inorganic mercury would be 6 $ng \cdot L^{-1}$ and 14 $ng \cdot L^{-1}$, respectively. This theory has not been tested over a wide range of environmental conditions, and is based on uptake from the water column (shoots) rather than from sediments (roots). Furthermore, the experiments preceded the use of trace-metal clean collection and analysis techniques, and should be repeated.

6.2 Invertebrates

Mercury is accumulated by aquatic invertebrates directly from water (including porewater), and actively, through ingestion of contaminated food. It may be bound on the outer membranes or chitinous exoskeleton, absorbed within gut contents, incorporated into body tissues and excreted. Mercury bound to outer membranes or exoskeletons may be lost with moulting (Zauke 1977). Also, egg-carrying females have higher concentrations than other individuals because MeHg is readily transferred across lipid bilayers into eggs. As previously stated, MeHg is more likely than inorganic mercury to be absorbed and retained in tissue because of its affinity for sulphydryl groups and its slow rate of depuration (Saouter et al. 1993). The rate of MeHg uptake from water has been shown to be related to metabolic rate (Visman et al. 1995). Invertebrates tend to have a lower percentage of MeHg than fish because any forms of mercury taken up are either associated with gut contents or the exoskeleton rather than absorbed into tissue. Also, the sources of mercury to which they are exposed are comprised of relatively low percentages of MeHg.

Sediment-associated invertebrates or 'benthos' (e.g., chironomids, amphipods, mussels) generally contain more mercury than those in the water column (e.g., *Daphnia*). Mercury uptake by sediment-dwelling organisms is likely favoured by the combination of high mercury concentrations in their diet as well as in their physical environment (Parkman and Meili 1993). Some suggest that uptake for mussels changes from water to particulates depending on the concentration and composition of suspended particles (Gagnon and Fisher 1997). While laboratory experiments suggested water was the major route of mercury uptake for *Mytilus edulis*, under estuarine conditions, when inorganic Hg on suspended sediments was up to 80 times higher than dissolved Hg, the particulate pathway was dominant (King and Davies 1987). Malley et al. (1996) found that uptake of MeHg by the freshwater mussel, *Pyganodon grandis* was higher after reservoir flooding experiments when suspended particulate concentrations increased substantially.

Because predacious invertebrates belong to a higher trophic level, they will sometimes (but not always) bioaccumulate more MeHg than non-predaceous invertebrates (Parkman and Meili 1993). For example, a predatory mite, *Hydracarina* exhibited the highest bioconcentration of THg and MeHg at $2.5 \cdot 10^5$ and $12.6 \cdot 10^6$ times higher than water, respectively (BCF: $ng \cdot g^{-1}$ dw per $ng \cdot L^{-1}$). In the same lakes, the predacious *Chaoborus* spp. bioconcentrated mercury to lower levels than most non-predacious taxa (Back and Watras 1995). Tremblay (1998) reported the mean proportion of MeHg in 16 Québec lakes increased from detritivores (35-45%; dipterans, ephemeropterans, trichopterans) to predacious species (70-85%; notonectids, coleopterans, odonates).

The proportion of accumulated mercury taken up either from water or food depends on degree of contamination of each compartment, as well as on the chemical forms of mercury present. Saouter et al. (1993) measured the distribution of mercury and MeHg in a common mayfly, *Hexagenia rigida*, after a nine-day exposure in laboratory microcosms containing both water and sediment. Mercury was added either to the overlying water or to the sediment. Methylmercury was taken up 60 times more readily than $HgCl_2$ when it was added to sediments, and twice as readily when added to overlying water. Both forms of mercury tended to accumulate in the gut when taken up through ingestion of sediments, whereas they accumulated on the gills when taken up from water (Saouter et al. 1993). Crayfish that were caged with MeHg contaminated or uncontaminated food and water, absorbed virtually all of their MeHg from their diet and not from water (Parks et al. 1988). Invertebrates usually have mercury body burdens of less than 65% MeHg because 1) inorganic mercury concentrations in sediments, particulates and water are proportionally higher than MeHg, and 2) because of the shorter life spans of invertebrates that limits the potential absorption of MeHg into tissue (Parkman and Meili 1993).

Even though MeHg is proportionately less in invertebrates than it is in fish, the trophic transfer of MeHg is much more efficient than for inorganic Hg (62% and 15%, respectively). Mason et al. (1995) demonstrated this principle experimentally between zooplankton and an algal food source by finding that the outer membrane and cytoplasm contained 37% and 63% MeHg, respectively; and 91% and 9% inorganic Hg, respectively. Since zooplankton digest the cytoplasm but defecate the membrane material, MeHg becomes biomagnified at lower levels of the food web because the bulk of the digested material contains a high proportion of MeHg.

Water quality variables can influence the uptake of mercury by aquatic invertebrates. For zooplankton, total mercury content varies positively with mercury concentration in water (Sorensen et al. 1990). For benthic invertebrates (as with aquatic plants), mercury concentrations are not correlated with sediment mercury concentrations (Kristensen 1982; Parkman and Meili 1993). Higher concentrations of MeHg in zooplankton have been reported in acidified lakes (Bloom 1992), especially if they were also brown-water (high DOC) drainage lakes (Westcott and Kalff 1996). Laboratory experiments confirmed that increased uptake of $HgCl_2$ and MeHgCl occurred with decreasing pH (Saouter et al. 1993) and that the diffusional fluxes of mercury over lipid bilayers were higher at pH 5.0 than at pH 8.5 (Boudou et al. 1991). There is some evidence that DOC suppresses uptake of both inorganic Hg and MeHg by invertebrates based on results that show a negative correlation of all forms of Hg in zooplankton with DOC in seepage lakes (no terrestrial sources of Hg; Watras et al. 1994; Back and Watras 1995).

Higher mercury concentrations are found in invertebrates that reside in the deepwater compared to shallow sediments, particularly in anoxic conditions (Särkkä 1979). Mercury concentrations may be ten times higher in deepwater than near shore taxa (Parkman and Meili 1993). The sediment-water interface is the zone of highest Hg concentrations, steep oxygen gradients, and high bacterial activity (thus methylation potential), therefore minor differences in feeding strategy may result in large differences in Hg bioaccumulation.

Invertebrates that feed on plants and sediments may contain more THg than do predatory invertebrates (Särkkä 1979; Parkman and Meili 1993). This is attributed to the high proportion of inorganic mercury found in non-predatory animals; predators would not accumulate inorganic mercury because it is poorly retained compared with MeHg (Parkman and Meili 1993). Theoretically, the larger body size of predators may reduce the mercury concentration in their tissue by growth dilution (important for fish). More research is required in this area.

As animals take up mercury through both diet and water, it is difficult to separate a bioconcentration factor, which is the concentration of a contaminant in the animal relative to the water concentration (BCF=[animal]/[water]), from a bioaccumulation factor (BAF), which theoretically includes uptake from both sources (i.e., [animal]/[water + food]; Metcalfe 1986). Whenever values are calculated from field results, a BCF is often reported, although the two uptake routes cannot truly be separated. When diet is reported to be the predominant route of uptake, comparing the contaminant concentration in the consumer to the concentration in their food is more representative. For example, crayfish had BAFs ([crayfish]/[diet]) of 4.5 to 8.3 when fed a high and low MeHg diet of fish tissue over a period of ten weeks (Parks et al. 1988). Some older examples of wet weight-based total mercury BCFs are 900 for a dragonfly nymph from a contaminated lake in Ontario (Clay Lake, Smith et al. 1975) and 29 000 for a dragonfly nymph from a stream in South Carolina (Cox et al. 1975)[8].

Because of the scarcity of MeHg measurements done on water, plants and invertebrates within individual studies, few bioconcentration factors and/or bioaccumulation factors for MeHg are available for invertebrates. Tremblay (1998) found a MeHg biomagnification of about three between the two adjacent trophic levels of phytoplankton and zooplankton. BCFs for zooplankton were reported to increase by 10 to 100 times more for MeHg compared to inorganic Hg (based on wet weights; Watras and Bloom 1992). Concentrations of MeHg and THg were higher in all compartments in the experimentally acidified basin (pH 4.7) of Little Rock Lake. The BCFs ([zooplankton]/[water]) were an order of magnitude higher for MeHg than for inorganic Hg. BAFs ([zooplankton]/phytoplankton]) were the same for inorganic Hg in both the acidified and reference basins (BAF=1.9); BAFs for MeHg were 3.6 and 5.0 in the acidified and reference basins, respectively (Watras and Bloom 1992). These data supported the hypotheses that the uptake of MeHg by zooplankton was more efficient than inorganic Hg, and that uptake of MeHg is proportional to supply regardless of lake acidification.

6.3 Freshwater Fish

Essentially all mercury found in piscivorous freshwater fish tissue is MeHg (approximately 99%). Early studies that reported less than 95% MeHg may have been limited by their analytical methodology or problems with homogeneity of subsamples (Bloom 1992). While inorganic mercury is always more abundant in natural ecosystems, it is absorbed by fish much less efficiently than MeHg, and if taken up, is eliminated more rapidly than is MeHg (Huckabee et al. 1979; Boudou and Ribeyre 1985; Trudel and Rasmussen 1997). This is because the digestive wall is much more permeable for MeHg than for inorganic mercury (Boudou and Ribeyre 1985), allowing MeHg to be readily transferred to other tissues.

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[8] Note that these BCFs are based on measurements pre-dating modern collection and analytical techniques.

Biomagnification of MeHg through diet, rather than gill uptake from water, is considered the dominant basis for elevated concentrations in fish (perhaps 90%; Harris and Snodgrass 1993; Rodgers 1994). Recent measurements of mercury speciation in water indicate fish BCFs for MeHg of 10^6 to 10^7, and for non-MeHg BCFs are less than 10^4 (Bloom 1992). This demonstrates that MeHg biomagnifies in fish by 100 to 1000 times more than other forms of mercury (compared to 10 to 100 times for zooplankton).

Harris and Snodgrass (1993) modelled the uptake of MeHg from water in yellow perch *(Perca flavescens)* and walleye *(Stizostedion vitreum)*, as well as uptake through two types of diet. Methylmercury concentrations were modelled with realistic concentrations for uncontaminated water ($0.05\ ng{\cdot}L^{-1}$), for zooplankton ($0.033\ ng{\cdot}g^{-1}$ ww) and for forage fish ($0.30\ ng{\cdot}g^{-1}$ ww). Ninety-nine percent of MeHg was accumulated from the diet over several years, taking about one year to reach equilibrium when the diet changed from zooplankton to forage fish. Even when the water concentrations were increased to 'unrealistically high' levels ($0.3\ ng{\cdot}L^{-1}$ MeHg), and allowing for uncertainty factors, uptake from water was always less than 20% of the total.

Their simulations indicated that excretion was more effective in young fish than from older fish, suggesting that remedial actions may be more effective in reducing mercury in younger fish (may take 10 years to excrete 50% for a 10-year-old walleye; Harris and Snodgrass 1993). The calculations were based on an assumed allometric exponent of -0.6 for Hg elimination. Trudel and Rasmussen (1997) recently calculated the exponent to be closer to -0.2, which would mean that smaller fish excrete MeHg more slowly than the Harris and Snodgrass model predicts. In general, MeHg is excreted very slowly relative to uptake for most fish. Methylmercury is not eliminated rapidly enough to prevent a net accumulation as long as exposure is continued.

Even in the absence of trophic transfer of MeHg, large adult predatory fish would be expected to have higher concentrations of MeHg than their prey because of their longer exposure as a result of age (predatory fish have longer life spans than forage fish) (Huckabee et al. 1979). Fish length, the preferred surrogate for age, is the best single predictor of Hg concentrations in fish (Richardson 1994; G.M. Richardson 1999, O'Connor Associates, pers. com.). Other factors such as differences in food consumption rates, food conversion efficiencies, and fish metabolic rates are also important (Canada-Manitoba Mercury Agreement 1987). The assimilation efficiency of MeHg from food to fish is often estimated at 80% (Norstrom et al. 1976; Harris and Snodgrass 1993; Trudel and Rasmussen 1997).

Recent information indicates that generalisations about the trophic level that a particular species occupies may lead to errors in predictions about potential MeHg concentration. For example, fish may be piscivorous in one lake, but insectivorous or omnivorous in another if no smaller forage fish occupy the lake. This difference in diet may cause the wholly piscivorous fish to have high mercury levels, while the same species in the lake with the shorter food chain may not. Cabana et al. (1994) clearly established that the food chain structure in 81 Ontario lakes had this effect for mercury accumulation by lake trout. Mercury levels in trout from lakes with the longest food chains where pelagic forage fish and the crustacean *Mysis relicta* were present were about 3.6-fold higher than those from lakes with the shortest food chains, where the two trophic components were missing. The importance of food chain length has been demonstrated for other persistent contaminants such as PCBs (Ontario lakes; Rasmussen et al. 1990) and toxaphene (Arctic lakes; Kidd et al. 1993). The relatively inexpensive procedure of measuring stable nitrogen isotopes may be used for screening the trophic level of fish from lakes prior to undertaking measurements of contaminants that biomagnify (Cabana and Rasmussen 1994).

Reservoirs may have fish with elevated mercury concentrations whether or not they have high pH or alkalinity[9]. In reservoirs, the flooded organic matter and associated increased bacterial activity enhances MeHg production (Canada-Manitoba Mercury Agreement 1987; Ramlal et al. 1987). Any bioavailable MeHg may then be bioaccumulated. Overviews of the mercury problem in specific reservoirs are found in Bodaly and Hecky (1979), Verta et al. (1986), Jackson (1987), Canada-Manitoba Mercury Agreement (1987), Verdon et al. (1991), Anderson et al. (1995), Rodgers et al. (1995), Verdon and Tremblay (1998), and a model of fish mercury levels as related to physical characteristics of Canadian reservoirs was developed by Johnston et al. (1991). While these reports concern reservoirs in Manitoba, Saskatchewan, Ontario, Québec, Labrador and Scandinavia, reservoirs in Alberta have not been associated with elevated mercury in fish to date (AEC 1989-Gleniffer L, Dickson Dam.; AEC 1993-Oldman River Dam). Screening for mercury in fish from reservoirs in British Columbia is in progress (Watson 1992).

Numerous chemical and physical variables of surface waters determine the potential for fish bioaccumulation of mercury (Table 7). In particular, low pH (<6), low alkalinity (acid-neutralising capacity 50 $\mu eq \cdot L^{-1}$ or less), and low calcium (<5 $mg \cdot L^{-1}$) lakes are associated with elevated mercury concentrations in fish (Grieb et al. 1990; Spry and Wiener 1991). Studies of the effect of pH on uptake by fish show conflicting results. In controlled laboratory studies, Rodgers et al. (1987) found no difference in accumulation of MeHg by walleye and trout among three pH treatments (5, 6, and 7); whereas Drummond et al. (1974) found greater MeHg accumulation by brook trout at pH 6 than 9. In drainage lakes (i.e., those that receive terrestrial runoff), DOC more strongly (positively) influences MeHg concentrations in fish than either pH or alkalinity, possibly through DOC-mediated transport of MeHg from the watershed[10] (Richardson 1994). DOC input into lakes is, in turn, negatively correlated to sulphate deposition in the drainage basin because the solubility of organic carbon declines as pH lowers. Thus, in drainage lakes, acidification may indirectly reduce MeHg levels in fish by limiting DOC-mediated transport of MeHg from the drainage basin (Richardson 1994). In contrast, acidification of seepage lakes may increase MeHg levels in fish because methylation is promoted at low pH (Miskimmin et al. 1992; Richardson 1994).

The efficiency of MeHg uptake across the gills of fish is inversely correlated with the calcium ion content of water (Rodgers and Beamish 1983). The water hardness relationship (calcium) has particular relevance in Alberta, where many lakes and rivers are high in calcium and pH. This may explain why fish in many Alberta reservoirs do not have elevated mercury concentrations even though other water bodies in western Canada do. Uptake would be reduced in these high calcium, high pH water bodies compared with softwater lakes. Water hardness makes little difference to mercury toxicity (Keller and Zam 1991). Selenium (Se) may effectively block the accumulation of MeHg in soft, as well as harder, more alkaline waters (Southworth et al. 1994). Accumulation of MeHg by fish in temperate waters is most rapid during summer when the feeding and metabolic rates of fish are most rapid and when the production of MeHg is greatest (Spry and Wiener 1991).

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[9] High pH and alkalinity are often associated with low Hg levels in fish.

[10] In seepage lakes that have little terrestrial runoff, DOC is less important than pH and alkalinity, and is negatively correlated with MeHg concentrations in fish presumably by inhibiting biotic methylation (Richardson 1994).

Table 7. Variables associated with mercury concentrations in fish

Taxa	Location	Fish [Hg] related to	Reference
Yellow Perch (Yearling)	Muskoka-Haliburton, ON	pH(-), $A_{dr.bas.}/A_{lake}$(+)	Suns & Hitchin 1990
Perch, Pike, Sucker, Bass	Michigan (UP)	pH(-), alkalinity(-), DOC in seepage lakes only (-)	Grieb et al. 1990
Yellow Perch (2 years old)	Wisconsin	pH(-), profundal sediment [Hg](+)	Cope et al. 1990
Walleye	79 Ontario lakes	DOC(+), Fe(+)	Wren et al. 1991
Northern Pike	79 Ontario lakes	DOC(+), Fe(+), pH(-), alkalinity(-)	Wren et al. 1991
Sunfish	16 South Central Ontario Lakes	pH(-)	Wren & MacCrimmon 1983
Yellow Perch	6 N. Ont. Shield lakes	Lake size (-); Epilimnetic temperature (+)	Bodaly et al. 1993
Pike	Sweden	pH(-), sed. [Hg](+), prox. To emission sources (+)	Håkanson et al. 1988
Lake trout, Sucker, Perch, Pike, Walleye	14 Ontario lakes	Fish weight(+), atmospheric Hg loading(+)	Johnson 1987
Yellow Perch (3-5 years)	16 Adirondack lakes	DOC(+), % wetlands(+), total dissolved Al(+)	Driscoll et al. 1994
Yellow Perch - MeHg	7 Wisconsin seepage lakes	BCF:DOC(-)	Watras et al. 1994
Yellow Perch - THg	7 Wisconsin seepage lakes	BCF:pH(-)	Watras et al. 1994

6.4 Accumulation by Semi-Aquatic Animals and Birds

Semi-aquatic animals and birds that consume aquatic biota bioaccumulate MeHg from their diet, similar to fish. Piscivorous mammals like mink and otter, and birds such as loons, herons and osprey, are top predators in ecosystems and may bioaccumulate substantial MeHg from their diet. Some birds, like the American wigeon (*Anas americana*), tend to accumulate less MeHg because of a diet consisting mostly of aquatic plants (Vermeer et al. 1973). The mechanism for transfer of inorganic Hg and MeHg from the gut to other tissues is the same as described for fish (section 6.3).

Organisms at higher trophic levels also tend to live longer, have more complex physiology and more organs that MeHg will accumulate in. Researchers usually find a lower proportion of MeHg in liver and kidney than in other tissues. While demethylation has been suggested as a mechanism for low MeHg in these organs (Wren et al. 1986), other possibilities may include tissue redistribution or selective excretion.

Mercury levels in mink and otter are elevated relative to the fish or other aquatic biota they consume. Kucera (1983) found that THg was an order of magnitude higher in mink and otter than in fish from the same river system in Manitoba. Somewhat lower average BAFs of 3.9 and 3.4 for mink and otter, respectively, were reported in a wide-ranging study of fish and mustelid populations in New York state (Foley et al. 1988).

Mercury may be passed from adult females to their offspring. Almost 100% of mercury transferred from adult loons through eggs to chicks was organic with no net loss of MeHg from chick tissue as the embryo developed. Methylmercury levels in eggs and in the brain of newly hatched chicks often exceeded levels in the mother's brain (Barr 1986). The transfer of Hg to eggs has been suggested as a method of Hg excretion (Heinz and Hoffman 1998). DesGranges et al. (1998) studied ospreys *(Pandion haliaetus)* in northern Québec and found that adults had higher total mercury concentrations in feathers than their fledglings and that there was no difference in the number of young that fledged per active nest between those located near reservoirs and those located near natural lakes and rivers.

Selenium has long been considered to be a protective agent against mercury toxicity (Wren 1984), a factor that will be discussed further in chapter 8 (Toxicity). Se may also be one factor that reduces MeHg accumulation according to the unexpected finding of low mercury in the tissues of mink and otter near Sudbury compared to other locations in Ontario. The mammals from the metal-contaminated Sudbury environment contained high levels of selenium (Wren and Stokes 1988). Mercury levels in fish from the Sudbury region were also lower than expected (Scheuhammer and Blancher 1994), shifting the potential selenium effect at least one step down on the food web in this case. High levels of sulphide would also be found in the Sudbury area. The solubility products (K_s) of HgSe and HgS are 10^{-58} and 10^{-52}, respectively (WHO 1990), which would indicate a strong binding of free mercury ions and a reduction of availability for methylation and accumulation. The Hg-Se ratio has shown an inconsistent tendency to be higher in freshwater fish than in marine fish, which may be an indication of the higher relative mercury levels in certain freshwater environments (Pelletier 1985).

Feather moult, exposure time and residual tissue levels from one season to the next for migratory piscivorous birds are important determinants of mercury body burdens. Hg concentrations were found to decrease in Bonaparte's gulls (*Lacus philadephia*) as moult progressed over the season (Braune and Gaskin 1987). Loons may spend a summer on a mercury-contaminated territory, then undergo a complete moult including renewal of primary feathers before migrating to winter breeding grounds (Barr 1986). Growing feathers may concentrate mercury up to 12 and 22 times more than breast muscle in adult and juvenile pintail ducks, respectively (Vermeer and Armstrong 1972). In loons, both back and belly feathers contained five to six times the mercury levels in breast muscle. Back feathers of adult loons collected in Ontario from 1971 to 1974 contained an average of 3.8 to 13.4 $mg{\cdot}kg^{-1}$, belly feathers contained 10.7 to 14.9 $mg{\cdot}kg^{-1}$, while breast muscle contained 1.02 to 3.26 $mg{\cdot}kg^{-1}$ (Frank et al. 1983). Thus, individual birds that winter on relatively mercury-free waters, should arrive on the breeding grounds with internal mercury levels lower than at any other time of year.

Methylmercury is preferentially retained in dark muscle rather than white muscle fibre of birds (c.f. Barr 1986). Dark and myoglobin-rich muscle fibre is characteristic of loons. Any residual tissue loads from either the previous season or contaminated winter habitats may be augmented sufficiently by contaminated prey (0.35 to 0.50 $\mu g{\cdot}g^{-1}$) in time to interfere with reproductive behaviour. For example, no loons hatched chicks in territories with yellow perch that had mercury levels exceeding 0.36 $\mu g{\cdot}g^{-1}$, while only one egg was laid in lakes from which mean Hg levels in perch exceeded 0.4 $\mu g{\cdot}g^{-1}$ (Barr 1986). These observations were from the mercury contaminated area of the English-Wabigoon River system in north-west Ontario. Other toxic effects will be discussed in Chapter 8.

7.0 OVERVIEW OF DISTRIBUTION AND LEVELS OF METHYLMERCURY IN THE CANADIAN ENVIRONMENT

This chapter describes methylmercury concentrations in aquatic plants, aquatic invertebrates, fish, amphibians, reptiles, semi-aquatic mammals, marine mammals, aquatic birds and non-biological media. Because of the quantity of measurements, note that the information contained in this chapter and tables are examples only and are not exhaustive. Instead, readers are referred to such databases as are being developed by Environment Canada (Kent et al. 1998), and to those that have been compiled for many years by some of the provinces (e.g., Ontario and Québec). Where MeHg concentrations for Canada are unavailable or scarce, information is supplemented with total mercury (THg) concentrations. In the case of fish, researchers routinely only measure THg because it is believed that essentially all mercury in fish tissue is MeHg. Each section is supported by detailed tables contained in Appendix B, with the number listed next to each sub-heading.

7.1 Aquatic Plants (App. B-1)

With a few exceptions, the available data indicate that methylmercury usually represents less than 50% of total mercury in freshwater aquatic plants. One study in Heney Lake, Ontario reported levels of 80 to 90 µg·kg^{-1} THg[11], and 50 µg·kg^{-1} MeHg in two genera of filamentous algae *(Mougeotia* and *Spirogyra)*, representing 55% MeHg (Stokes et al. 1983). In another lake from the same study (Swan Lake), MeHg represented less than 50% of THg from the same algal groups.

A recent study completed at the Experimental Lakes Area (near Kenora, ON) found that the percentage of MeHg depended on the type of tissue from *Sphagnum augustifolium* that was measured. The lower stem, upper stem, and capitulum contained 0.3%, 1.4% and 2.7% MeHg, respectively (0.3, 1.6, 2.5 µg·kg^{-1} dw; Moore et al. 1995). They also found that the type of habitat appeared to affect the levels of MeHg in *Sphagnum* spp. Species from a pool/stream habitat contained about 20 µg·kg^{-1} (19%) MeHg, while species from hollow/lawn and hummock habitats contained about 1 and 0.5 µg·kg^{-1}, respectively (2.5% and 1.4% MeHg). The macrophyte and sedge species, *Carex aquatilis, C. rostrata* and *Sparganium* spp. contained about 2 µg·kg^{-1} MeHg, on average, representing 37% of THg (Moore et al. 1995).

Macrophytes from the Ottawa River contained lower percentages of MeHg in their roots than their shoots. The roots of *Elodea canadensis, Sagittaria latifolia, Sparganium augustifolium,* and *Sparganium eurycarpum* contained 8 to 14% (mean = 10.3%) MeHg compared with 23 to 48% (mean = 31.7%) MeHg in their shoots (Mortimer 1985). The levels of THg were 48 to 550 µg·kg^{-1}, while the specific MeHg levels were not reported. The highest level of total mercury in aquatic plants found in the literature was 1600 µg·kg^{-1} (min. = 17µg·kg^{-1}), in *Myriophyllum* spp. collected in 1978 from the St. Lawrence River, near Cornwall (Mortimer 1985). *Myriophyllum* from the same area of the St. Lawrence River in 1994 contained THg concentrations in the range of 63 to 240 µg·kg^{-1} (Thompson-Roberts et al. 1999).

No data were located on levels of methylmercury in estuarine or marine plants in the Canadian environment.

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[11] All plant measurements on a dw basis unless noted otherwise.

7.2 Aquatic Invertebrates (App. B-2)

Many measurements of THg in aquatic invertebrates in Canadian freshwater environments have been made (App. B-2a), but it was not until recently that researchers began to report MeHg in invertebrates. Generally, the proportion of MeHg in invertebrates is higher than in aquatic plants.

In a wide-ranging study in northern Québec, Tremblay et al. (1996) found from 6 to 100% of THg in benthic invertebrates was MeHg. The two highest measured levels of MeHg were found in corixids (*Sigara* spp.) in the La Grande-2 Resevoir. In the south arm of the reservoir, the MeHg concentrations in the invertebrates averaged 1.52 $\mu g \cdot g^{-1}$ dw (representing 91% of THg) and in the north arm levels averaged 1.37 $\mu g \cdot g^{-1}$ (representing 76% of THg). Larval dragonflies (*Somatochlora* spp.) in the south arm contained 100% MeHg, with levels averaging 0.62 $\mu g \cdot g^{-1}$, while for the same organism in the north arm, only 75% of THg was MeHg. In a control lake, Duncan L, concentrations in various benthic invertebrates ranged from 0.014 $\mu g \cdot g^{-1}$ (mayfly, *Leptophlebia* spp.), to 0.124 $\mu g \cdot g^{-1}$ (corixid, *Sigara* spp.), with the proportion of MeHg ranging from 11% to 75%. See Appendix B-2 for specific results of this survey and Tremblay (1998) for further discussion of MeHg levels in invertebrates from lakes and reservoirs in northern Québec.

A study of twenty-four lakes in south-central Ontario lakes reported *Daphnia* species to contain 0.019 to 0.448 $\mu g \cdot g^{-1}$ dw MeHg (Westcott and Kalff 1996). They did not measure THg, so the proportion represented by MeHg could not be calculated (K. Westcott 1999, pers. com.).

Malley et al. (1996) found that while the kidney of the mussel, *Pyganodon grandis*[12] contained higher concentrations of MeHg than other tissues, the proportion of MeHg in the kidney was the lowest, at 15%; of 1.99 $\mu g \cdot g^{-1}$ dw THg in the kidney, 0.29 $\mu g \cdot g^{-1}$ was MeHg. The whole organism contained 44% MeHg, while the mantle, viscera, gill and foot contained 45%, 47%, 58% and 62% MeHg, respectively. This represented a range of 0.11 to 0.25 $\mu g \cdot g^{-1}$ MeHg (Malley et al. 1996). All mussels were from an unmanipulated source lake (Lake 104) at the Experimental Lakes Area, northern Ontario.

Organisms from the contaminated Wabigoon River/Clay Lake area of north-western Ontario showed among the highest levels of THg in freshwater invertebrates. For example, the crayfish, *Orconectes virilis*, from the east basin of Clay Lake collected in 1979-80 contained 2.2 ± 0.66 $\mu g \cdot g^{-1}$ ww THg, and from the Wabigoon River (inflow to Ball Lake) contained 1.7 ± 0.49 $\mu g \cdot g^{-1}$ ww THg (Parks et al. 1991). These are high levels for invertebrates considering that the consumption guideline for human consumers of fish in Canada is 0.5 $\mu g \cdot g^{-1}$ ww[13]. If the levels for the crayfish were converted to a dry weight basis, they would be even higher. For example, if tissues were considered at least 50% water, THg concentrations would be twice as high as reported above.

There are relatively few studies in Canada that look at mercury concentrations in marine invertebrates (App. B-2b). Concentrations of THg in the estuarine mussel, *Mytilus edulis*, were reported in areas of the St. Lawrence Estuary for the year of cessation of chlor-alkali plant activities and one year later (1976-1977). Concentrations at the mouth of Saguenay Fjord declined from 0.63 $\mu g \cdot g^{-1}$ dw to 0.17 $\mu g \cdot g^{-1}$ dw during this period. Similarly, levels of THg declined in two locations on the south shore of the lower estuary and in the Gaspé Peninsula during the same period (Cossa and Rondeau 1985; Appendix B-2).

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[12] Formerly *Anodonta grandis grandis*

[13] To be discussed in more detail in chapter 9.

7.3 Fish (App. B-3)

As previously described, mercury in fish from natural environments is comprised almost exclusively of MeHg regardless of the composition of diet sources and exposure to water. As such, the data tabulated includes numerous measurements of THg only, which are assumed to be MeHg concentrations unless specifically reported as measured MeHg. While the data included are not all-inclusive, information on freshwater fish mercury levels from eight provinces (Alberta, British Columbia, Manitoba, New Brunswick, Newfoundland, Nova Scotia, Ontario, Québec) and one territory (Northwest Territories) are included.

In some cases, water bodies were either directly contaminated with mercury by mine tailings or industry, and in other cases, MeHg levels became elevated as a result of hydroelectric reservoir development, or by high geologic levels of mercury. As expected, levels are generally highest in piscivorous species such as lake trout, northern pike, walleye and bull trout. The literature is dominated by reports on freshwater fish in comparison to marine fish. All levels are reported as $\mu g \cdot g^{-1}$ wet weight. Age and/or size class of the fish are tabulated where known; wildlife typically consume fish <35 cm in length (US EPA 1993). Results are discussed in alphabetical order by province or territory.

Monitoring programs in Canada typically measure mercury levels in large, commercial or sport fish to address human health concerns. Larger (i.e., older) fish, regardless of other influences, are expected to contain higher levels of mercury because of their longer exposure period relative to smaller (i.e., younger) fish consumed by wildlife. As such, levels reported below may be higher than those expected for prey species of wildlife. Care must be taken when interpreting the relevance of data for large fish to wildlife. The reader is cautioned against generalising the complex relationship between fish size and mercury concentration. While fish size has been shown to be the single best predictor of mercury body burdens, estimating mercury concentrations in small fish from measured concentrations in large fish depends on species and trophic level as well as on site-specific environmental (e.g., pH, alkalinity, DOC, basin/lake morphology) and physiological (e.g., uptake/elimination rates, assimilation efficiency) factors (see chapter 6).

7.3.1 Freshwater fish (App. B-3a)

In Alberta, high levels of mercury in fish were reported from lakes contaminated with mine tailings. Lake trout *(Salvelinus namaycush)*, northern pike *(Esox lucius)*, and round whitefish *(Prosopium cylindraceum)* collected in 1977 from Giauque Lake in northern Alberta contained averages of 3.79, 1.75 and 1.22 $\mu g \cdot g^{-1}$ ww, respectively (Moore and Sutherland 1980). In another contaminated lake, Thompson Lake, northern pike collected in 1978 had an average of 1.7 $\mu g \cdot g^{-1}$ THg in muscle tissue. Regardless of whether a lake was contaminated or not, lake whitefish *(Coregonus clupeaformis)* had low average mercury concentrations of 0.2 $\mu g \cdot g^{-1}$. In a number of control lakes, lake trout and northern pike muscle concentrations ranged from 0.11 to 1.0 and 0.05 to 1.91 $\mu g \cdot g^{-1}$, respectively (Moore and Sutherland 1980).

Ramamoorthy et al. (1985) reported mercury levels in seven fish species from the North Saskatchewan River in the vicinity of Edmonton. Average concentrations ranged from 0.245 $\mu g \cdot g^{-1}$ for longnose sucker *(Catostomus catostomus)* to 0.743 $\mu g \cdot g^{-1}$ for sauger *(Stizostedion canadense)*. Two other species that exceeded the consumption guideline of 0.5 $\mu g \cdot g^{-1}$ were goldeye (*Hiodon alosoides*; 0.594 $\mu g \cdot g^{-1}$) and walleye (0.645 $\mu g \cdot g^{-1}$).

Ramamoorthy et al. (1985) also reported the proportion of MeHg to range from 77% (longnose sucker) to 95% (walleye)[14].

In British Columbia, mercury in fish has been quantified in a number of lakes and rivers, as well as Williston Lake (reservoir associated with the Bennett Dam on the Peace River). Studies by BC Hydro on a number of other reservoirs are in progress (Watson 1992). The highest mercury concentrations were found in lake trout from Pinchi Lake, which is located in an area of high geological mercury and a former mercury mine. Mercury in these lake trout ranged from 1.06 to 5.78 $\mu g \cdot g^{-1}$ (Watson 1992). Bull trout (*Salvelinus confluentus*) levels were relatively high both in Pinchi Lake (0.49 to 0.75 $\mu g \cdot g^{-1}$) and in Williston Lake (0.25 to 1.62 $\mu g \cdot g^{-1}$). Kokanee *(Oncorhynchus nerka kennerlyi)* and rainbow trout *(Oncorhynchus mykiss)* from Pinchi Lake contained moderate to high levels at 0.32 to 0.76 $\mu g \cdot g^{-1}$ and 0.20 to 0.40 $\mu g \cdot g^{-1}$, respectively.

In a number of rivers in northern BC, mercury concentrations ranged from 0.02 to 0.78 $\mu g \cdot g^{-1}$. On average, rainbow trout and lake whitefish had the lowest levels, while burbot *(Lota lota)* and bull trout had higher levels (Watson 1992).

In Manitoba, extensive monitoring of mercury in fish has been done in association with reservoirs. Average mercury concentrations associated with the Churchill River diversion and flooding (Notigi Reservoir) including Southern Indian Lake exceeded 0.5 $\mu g \cdot g^{-1}$ in all species except lake whitefish (Bodaly et al. 1984[15]). Average mercury levels exceeded 2 $\mu g \cdot g^{-1}$ in walleye and northern pike from the Rat Lake portion of the Notigi Reservoir, and exceeded 1 $\mu g \cdot g^{-1}$ in most other areas of the Notigi Reservoir. Levels were also elevated (>0.75 $\mu g \cdot g^{-1}$) in these species in five lakes downstream of the Notigi Reservoir. In Southern Indian Lake, walleye and northern pike had average muscle concentrations of 0.57 $\mu g \cdot g^{-1}$ and 0.68 $\mu g \cdot g^{-1}$, respectively, while lake whitefish averaged 0.13 $\mu g \cdot g^{-1}$ (Bodaly et al. 1984).

Relatively fewer data are available for freshwater fish in the Atlantic provinces, although the dataset has recently grown because of a concern for loon populations in some areas. In a multi-agency study in Kejimkujik National Park, NS, hundreds of measurements in fish tissue have been made (report publications pending, Mercury Team 1998). Two land-locked salmon *(Salmo salar)*[16] from Sisson Branch Reservoir in the St. John River basin (NB), measuring 38 and 58 cm and weighing less than 1.5 kg, contained 0.84 and 1.5 $\mu g \cdot g^{-1}$ THg, respectively (Bailey 1985). Twelve lake whitefish from the Green River Reservoir (NB) averaged 0.12 $\mu g \cdot g^{-1}$. In the Smallwood Reservoir in Labrador during the 1970s, land-locked salmon reportedly contained 1.5 to 2.3 $\mu g \cdot g^{-1}$ (Bruce and Spencer 1978, c.f. Bailey 1985). Mercury measured in 1977 and 1978 in northern pike from Labrador lakes ranged from 0.01 to 3.08 $\mu g \cdot g^{-1}$ (n=117), in lake trout from 0.12 to 3.37 $\mu g \cdot g^{-1}$ (n=107) and in brook trout from 0.01-1.69 $\mu g \cdot g^{-1}$ (n=96; Bruce et al. 1979).

In Wreck Cove Reservoir, NS, brook trout weighing from 49-211 g had muscle mercury concentrations of 0.27-0.73 $\mu g \cdot g^{-1}$, averaging 0.50 ± 0.16 $\mu g \cdot g^{-1}$ (ADI Nolan Davis Inc. 1994). The anadromous striped bass *(Morone saxatilis)* in the Annapolis River had elevated Hg in muscle tissue ranging from 0.26 - 1.97 $\mu g \cdot g^{-1}$ (mean 0.77, mean weight of 7.4 kg); lower for smaller specimens (mean 1.8 kg) from the Shubenacadie River ranging from 0.16 - 1.44 $\mu g \cdot g^{-1}$ (mean 0.40 $\mu g \cdot g^{-1}$; Ray et al. 1984).

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[14] Note that recent methods and findings stress that 98-100% of THg in fish muscle is MeHg (Bloom 1992).

[15] Others have reported on mercury in these systems; the summary of Bodaly et al. 1984 is used for brevity here.

[16] Species not reported, Atlantic salmon is professional judgement of species based on location.

In the Northwest Territories, mercury concentrations in a variety of species averaged below 0.5 $\mu g \cdot g^{-1}$, with some exceptions. Lake trout from Kaminak Lake, west of Hudson Bay, contained 0.57 to 2.0 $\mu g \cdot g^{-1}$ THg (Shilts and Coker 1995). The Kaminak Lake area has high geological mercury. For unknown reasons, northern pike, lake trout and walleye have elevated mercury (means of 0.74, 1.34, 1.49 $\mu g \cdot g^{-1}$) in Lac Ste. Thérèse in the Johnny Hoe River system (Stephens 1995). The high levels have persisted over time, with measurements in walleye being made in 1975, 1980, 1992 and 1993. Walleye, but not pike or lake whitefish have average concentrations that exceed 0.5 $\mu g \cdot g^{-1}$ in another lake in the system, Tseepantee Lake. Two additional lakes (Keller and Taché) have fish with relatively lower THg concentrations.

In the Hay River, NWT, lake whitefish, walleye and northern pike averaged 0.07, 0.22 and 0.32 $\mu g \cdot g^{-1}$, respectively. Similar levels were found for these species in the Slave River and in Leland Lake (Grey et al. 1995).

More studies have been completed in Ontario for a wide range of water bodies than in most other regions of Canada. The Ontario Ministry of the Environment maintains a very large database of mercury concentrations in fish ("probably the largest in the world", G. Mierle 1999, MOE, pers. com.). The most highly mercury contaminated area in the country is likely the English-Wabigoon River system in north-western Ontario. The source of mercury was a chlorine-alkali plant located in the town of Dryden (Rudd et al. 1983). Very high levels of mercury in Clay Lake burbot and walleye were 21.9 and 15.7 $\mu g \cdot g^{-1}$ on average, respectively (Fimreite and Reynolds 1973). In Ball Lake, downstream of Clay lake, walleye, rock bass *(Ambloplites upestris)* and northern pike averaged 4.5, 6.2 and 7.7 $\mu g \cdot g^{-1}$. White suckers *(Catostomus commersoni)* from Clay Lake and the Wabigoon River had mercury levels in excess of 3 and 4 $\mu g \cdot g^{-1}$, respectively. Fish from many other connected lakes and rivers also had elevated mercury concentrations (Fimreite and Reynolds 1973).

Cabana et al. (1994) found a positive correlation between lake trout mercury concentrations and length of the food chain in large number of Ontario lakes. Lake trout from lakes with pelagic forage fish and *Mysis* shrimp had an average of 0.65 $\mu g \cdot g^{-1}$ compared with 0.18 $\mu g \cdot g^{-1}$ in lake trout from lakes that contained *Mysis* but lacked forage fish. The relationship did not exist for smallmouth bass *(Micropterus dolomieu)*, a species that does not exploit the pelagic food chain, for which THg averaged from 0.43 to 0.53 $\mu g \cdot g^{-1}$ regardless of the length of the food chain.

Lake trout had high mercury concentrations relative to rainbow smelt *(Osmerus mordax)* in a series of Ontario lakes examined from 1977 to 1981. Lakes Muskoka, Mary and Vernon were rated high mercury because lake trout concentrations averaged 2.96 to 4.49 $\mu g \cdot g^{-1}$ (0.34 to 0.49 for smelt). Lakes Koshlong, Rosseau and Tadenac were rated moderate because lake trout concentrations averaged 0.61 to 0.99 $\mu g \cdot g^{-1}$ (0.18 to 0.26 for smelt), and Lakes Simcoe, Joseph and Bella were rated low Hg because of lake trout concentrations of 0.39 to 0.55 $\mu g \cdot g^{-1}$ (0.08 to 0.14 for smelt; MacCrimmon et al. 1983).

Northern pike from 170 lakes and walleye from 255 lakes from across Ontario indicate that fish of a certain size from seemingly unpolluted systems overall average at or above the guideline level of 0.5 $\mu g \cdot g^{-1}$. Northern pike of approximately 54 cm in length averaged 0.50 $\mu g \cdot g^{-1}$ (0.07 to 1.28 $\mu g \cdot g^{-1}$), and walleye of approximately 41 cm in length averaged 0.58 $\mu g \cdot g^{-1}$ (0.09 to 3.24 $\mu g \cdot g^{-1}$) over a wide variety of lakes (Wren et al. 1991).

Yearling yellow perch collected both from the Ottawa River and a number of lakes in north-western Ontario had consistently low levels of mercury. Perch collected in 1974 and 1975 from the Ottawa River contained 0.13 $\mu g \cdot g^{-1}$ (Rodgers and Qadri 1982) and from six remote lakes from NW Ontario averaged 0.04 to 0.14 $\mu g \cdot g^{-1}$ (Bodaly et al. 1993). Similarly, yearling yellow perch collected from 16 lakes in the Muskoka-Haliburton area contained 0.031-0.233 $\mu g \cdot g^{-1}$ (Suns and Hitchin 1990).

In Québec, studies have focused on northern hydroelectric projects in the Great Whale, La Grande, and Nottaway-Broadback-Rupert (NBR) project areas. The highest levels were found in northern pike and walleye from the La Grande-2 Reservoir. Fish collected from 1982 to 1988 had average muscle concentrations of 1.31 to 2.99 $\mu g \cdot g^{-1}$ (pike), and 1.92 to 2.80 (walleye), with the highest averages occurring nine years after flooding (Verdon et al. 1991). Longnose sucker concentrations in the La Grande-2 reached a high of 0.67 $\mu g \cdot g^{-1}$ and lake whitefish a high of 0.57 $\mu g \cdot g^{-1}$ five years after flooding. Twenty-nine control lakes in the region indicated that fish accumulated lower amounts of mercury with concentrations in northern pike of 0.25 to 0.90, walleye of 0.32 to 1.26 and longnose sucker of 0.06 to 0.32 $\mu g \cdot g^{-1}$ (Verdon et al. 1991). More recent publications summarise fish mercury levels at the La Grande complex from 1978 to 1994 (Hydro-Quebéc and Groupe-conseil Génivar Inc. 1996, 1997).

In the Great Whale and NBR project areas of northern Québec, average mercury concentrations in piscivorous fish exceeded the 0.5 $\mu g \cdot g^{-1}$ level. Northern pike and walleye from the NBR region on the south-east coast of James Bay had levels of 0.93 ± 0.51 and 0.78 ± 0.46 $\mu g \cdot g^{-1}$, respectively. Mercury concentrations in these species were only slightly lower in the Great Whale area on the east coast of Hudson Bay (Langlois et al. 1995). Additional data for these regions may be found in Hydro-Quebéc and Groupe-conseil Génivar Inc. (1997).

Measurements of mercury in northern pike and walleye from 235 lake and 214 river sites across Québec indicated that average levels exceeded 0.5 $\mu g \cdot g^{-1}$ in many cases. Northern pike between 40-50, 55-70, and >70 cm measured 0.4, 0.66, and 1.12 $\mu g \cdot g^{-1}$, respectively. Walleye between 30-40, 40-50, and >50 cm averaged 0.53, 0.78, and 1.26 $\mu g \cdot g^{-1}$, respectively (Laliberté 1996).

7.3.2 Marine fish (App. B-3b)

A few data for methylmercury in marine fish in the Canadian environment will be discussed. Atlantic herring *(Clupea harengus harengus)* collected in 1981 from the south-western Bay of Fundy had low levels of mercury ranging from 0.005 to 0.015 $\mu g \cdot g^{-1}$. The fish were one to five years in age, and mercury in muscle and whole body was found to increase with age (Braune 1987a). Greenland cod (*Gadus ogac*, 400 mm) caught in the coastal waters of James Bay between 1987 and 1994 contained from 0.14 to 0.42 $\mu g \cdot g^{-1}$ of mercury, while the fourhorn sculpin (*Myoxocephalus quadricornis*, 250 mm) from the same regions contained from 0.1 to 0.55 $\mu g \cdot g^{-1}$ (Hydro-Quebéc and Groupe-conseil Génivar Inc. 1997).

7.4 Amphibians (App. B-4)

No data were located on levels of methylmercury in amphibians in the Canadian environment. One recent study examined levels of THg in the mudpuppy, *Necturus maculosus*, from various locations in the St. Lawrence and Ottawa Rivers. Average concentrations ranged from 0.087 to 0.239 $\mu g \cdot g^{-1}$ on a wet weight (ww) basis, with a maximum individual concentration of 0.445 $\mu g \cdot g^{-1}$ ww (Bonin et al. 1995). The high concentration was an adult female from the Beauharnois area of the St. Lawrence River; whole body with gonads removed. The average whole body concentration (without gonads) was 0.239 ± 0.137, while the gonads of the same six individual females averaged 0.068 $\mu g \cdot g^{-1}$ ww.

7.5 Reptiles (App. B-4)

No data were located on levels of methylmercury in reptiles in the Canadian environment. Snapping turtle eggs *(Chelydra serpentina)* collected in 1989 and 1990 from the St. Lawrence River and tributaries contained relatively low concentrations of THg (0.09 to 0.15 µg·g^{-1} ww THg; Bonin et al. 1995).

7.6 Mammals

7.6.1 Semi-aquatic Mammals (App. B-5)

No data were located on levels of methylmercury in semi-aquatic mammals in the Canadian environment. Several studies that report total mercury concentrations will be described briefly.

In a number of locations in Manitoba, mink *(Mustela vison)* and otter *(Lutra canadensis)* accumulated higher THg concentrations in liver tissue than in kidney or brain tissue. Average liver concentrations ranged from 0.82 to 3.93 µg·g^{-1} ww for mink, and from 1.75 to 3.67 for otter. Average kidney concentrations ranged from 0.67 to 2.98 µg·g^{-1} THg for mink, and from 1.07 to 2.37 µg·g^{-1} for otter. The maximum THg concentration in brain tissue was 0.97 µg·g^{-1} for mink, and 0.92 µg·g^{-1} for otter (Kucera 1983). The highest levels tended to be from animals in the vicinity of the Winnipeg River.

In Ontario, MeHg percentages for various tissues in mink and otter from the English River, Muskoka and Sudbury areas were reported. Mink livers and kidneys contained 53% and 43% MeHg, respectively, based on THg concentrations ranging up to 7.5 and 5.54 µg·g^{-1}, respectively. Otter livers, kidneys and muscle contained 34%, 19%, and 90% MeHg, respectively, based on THg concentrations ranging up to 17.4, 12.6, and 4.26 µg·g^{-1}, respectively (the highest concentrations were from the English River; Wren et al. 1986). Details of THg concentrations in all tissues are reported in Appendix B-5.

In an Ontario study of mink and otter, THg in liver, kidney and brain tissue showed similar trends as the Manitoba study of decreasing concentration with type of tissue. For mink, the highest average liver tissue concentration was 2.55 ± 1.96 (range of 0.56 to 6.9) µg·g^{-1} from the contaminated English River district of north-western Ontario. For otter, the highest average liver tissue concentration was 3.47 ± 4.1 (range of 0.71 to 17.4) µg·g^{-1} from the same area (Wren et al. 1986). Muscle concentrations in the province-wide study ranged from 0.38 to 1.62 µg·g^{-1} for mink, and 0.30 to 1.04 for otter.

In northern Québec, mink from the Great Whale area had the highest average muscle tissue concentrations of 2.40 ± 2.24 µg·g^{-1} THg (Langlois et al. 1995). Mink from natural environments of northern Québec had liver, kidney and brain tissue concentrations of 4.85, 2.45 and 0.93 µg·g^{-1} THg (Bélanger and Larivière 1997).

Other semi-aquatic mammals that have been monitored for THg include beaver *(Castor canadensis)* and racoon *(Procyon lotor)*. Liver, kidney and muscle tissue from beaver collected near Parry Sound, ON, contained 0.03 µg·g^{-1} THg. This low level reflects the non-fish diet of the beaver. Liver, kidney and muscle tissue from racoons collected in the same area contained 4.5, 1.1 and 0.3 µg·g^{-1} THg (Wren 1984).

7.6.2 Marine Mammals (App. B-6)

Few studies report on MeHg in marine mammals in the Canadian environment. Some of the highest THg levels found were measurements of marine mammal livers. The following summarises some of the elevated THg and MeHg levels (fully detailed in Appendix B-6) in marine mammal livers in many regions of Canada:

Species	THg $\mu g \cdot g^{-1}$	MeHg $\mu g \cdot g^{-1}$	Reference
Beluga whale (St. Lawrence R.) *(Delphinapterus leucas)*	33	NR	Wagemann et al. 1996
Ringed seal (Western Arctic) *(Phoca hispida)*	33	NR	Wagemann 1995
Harbour seal[17] (Great Whale Area) *(Phoca vitulina)*	29	4.6	Langlois et al. 1995
Pilot Whale (Atlantic Coast) *(Globicephala malaena)*	23	NR	Wagemann et al. 1995
Beluga whale (Great Whale Area)	20	7.3	Langlois et al. 1995
Narwhal (Eastern Arctic) *(Monodon monoceros)*	11	NR	Wagemann et al. 1996
Harp seal (Atlantic coast) *(Phoca groenlandica)*	10	NR	Wagemann et al. 1995

Livers of ringed seal *(Phoca hispida)* are consumed by indigenous people. Wagemann (1994) determined that the concentration of MeHg in ringed seal liver relative to THg was only 15% to 30%. He later argued that the MeHg percentage in ringed seal liver was much lower (only 2 to 3%) based on more accurate calculation methods (Wagemann et al. 1997; R. Wagemann 1999, DFO, pers. com.). The liver of ringed seals listed above were collected from the western Arctic between 1987 and 1993. Based on Wagemann et al. (1997) the concentration of MeHg in the above liver tissue averaged about 1.0 $\mu g \cdot g^{-1}$. Ringed seal livers from the eastern Arctic contained 28 $\mu g \cdot g^{-1}$ THg (3% = 0.8 $\mu g \cdot g^{-1}$ MeHg; Wagemann et al. 1995; R. Wagemann 1999, DFO, pers. com.), and from the Great Whale area, 1.1 $\mu g \cdot g^{-1}$ MeHg (Langlois et al. 1995).

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[17] Land-locked, described as freshwater seals.

There are spatial and temporal trends to mercury accumulations in Arctic marine mammals. In general, tissues of ringed seals and belugas from the western Arctic have higher mercury concentrations than those from the eastern Arctic. While this was attributed to different natural backgrounds of mercury in the two regions, it is important to note that 1) mercury in the liver of belugas increased in the western and eastern Arctic over 10 to 12 years; 2) mercury in the liver of ringed seals in the western Arctic and narwhal in the eastern Arctic showed similar increases; 3) belugas collected in the mid 1990s accumulated twice as much mercury as those collected 10 to 12 years ago; and 4) ringed seals collected in the mid-1990s accumulated three times more mercury than those collected 15 to 20 years ago (Wagemann et al. 1996).

The recent results of Wagemann et al. (1997) indicate that mercury in the muscle tissue of narwhal *(Monodon monoceros)*, ringed seal and belugas *(Delphinapterus leucas)* is essentially all MeHg. On the other hand, at least one report indicated that only 34% of THg in ringed seal muscle tissue was MeHg (Langlois et al. 1995). The residues were not excessive in either case, with levels of 0.6 $\mu g \cdot g^{-1}$ MeHg (Wagemann et al. 1997) in Arctic seals, and 0.11 $\mu g \cdot g^{-1}$ in seals from the Great Whale area (Hudson Bay; Langlois et al. 1995).

Belugas in the St. Lawrence River aged from young-of-the-year to 30 years contained an average of 33.6 $\mu g \cdot g^{-1}$ THg in liver tissue (maximum of 202 $\mu g \cdot g^{-1}$; Wagemann et al. 1996). This was the highest recorded average mercury concentration. Methylmercury levels were not reported. In muscle, belugas from the Arctic, St. Lawrence River, and Great Whale areas contained in excess of 2 $\mu g \cdot g^{-1}$ (Langlois et al. 1995; Wagemann et al. 1997). Since much of the mercury in marine mammal muscle tissue is MeHg, belugas from many areas of Canada can be considered to have excessive MeHg in muscle tissue.

7.7 Birds (App. B-7)

Numerous studies report THg in various bird tissues, however, relatively few report methylmercury concentrations. While some analyses indicate the majority of mercury in tissues except liver and kidney is MeHg, there is not enough evidence to conclude, as with fish, that all mercury in birds is MeHg. Fish-eating birds commonly have a high proportion of MeHg in muscle and brain tissue. Eggs contain essentially all MeHg, which is transferred from females prior to laying.

An early study on single specimens of immature birds in the vicinity of Clay Lake, north-western Ontario (mercury contaminated) found that 69% to 99% of THg in breast tissue was MeHg. The species examined were blue-winged teal *(Anas discors)*, common goldeneye *(Bucephala clangula)*, common merganser *(Mergus merganser)*, hooded merganser *(Lophodytes cucullatus)*, and mallard *(Anas platyrhynchos)*. The hooded merganser had the highest MeHg concentration with 17.8 $\mu g \cdot g^{-1}$ (99%), while the mallard had the lowest MeHg concentration with 0.11 $\mu g \cdot g^{-1}$ (69%). The teal, goldeneye and common merganser had 7.4, 12.3, and 14.9 $\mu g \cdot g^{-1}$, respectively (Vermeer et al. 1973).

Common loons in the Algonquin Park and Parry Sound areas collected from 1971 to 1974 had higher levels of mercury in those individuals that were emaciated. Muscle tissue contained 2.7 to 5.4 $\mu g \cdot g^{-1}$ in emaciated loons compared to 0.8 to 1.1 $\mu g \cdot g^{-1}$ in healthy loons (averages; Frank et al. 1983). Mercury concentrations were similarly approximately three times higher in the brain tissue of emaciated loons compared to healthy loons (Appendix B-7).

Another study undertaken in the 1970s involved an extensive examination of the common loon *(Gavia immer)* in the Wabigoon-English River district of north-western Ontario. This is the same vicinity as Clay Lake described above. Loon eggs contained 97% to 100% MeHg with the highest concentration of 1.34 ± 0.53 $\mu g \cdot g^{-1}$ in a series of six lakes downstream of Dryden on the Wabigoon River system (Barr 1986). Loon chick muscle, brain and liver contained 0.3 $\mu g \cdot g^{-1}$ (82-85%), 0.4 $\mu g \cdot g^{-1}$ (99%), and 0.8 $\mu g \cdot g^{-1}$ (87-92%) MeHg, respectively. Adult loon muscle, brain and liver contained 0.9 to 2.3 $\mu g \cdot g^{-1}$ (50-86%), 0.3 to 0.8 $\mu g \cdot g^{-1}$ (52-92%), and 0.2 to 1.5 (4-27%) MeHg, respectively (Barr 1986). The pattern seems to be that the young have mainly MeHg in their tissues, and the livers of adults do not favour MeHg accumulation over other forms of mercury (similar to marine mammals). Studies from other less contaminated areas are needed to corroborate this and other conclusions.

In a recent study in Ontario, both adult and chick loon blood mercury concentrations were positively correlated with fish mercury concentrations (r = 0.71 and 0.51, respectively; Scheuhammer et al. 1998). Three of twenty-four lakes had 20 to 50 g perch and sunfish with Hg concentrations exceeding 0.3 $\mu g \cdot g^{-1}$, the level reported to cause reproductive impairment in loons (Barr 1986). As reported by Evers et al. (1998) in their Wisconsin study, Scheuhammer et al. (1998) found that adults had blood mercury concentrations more than ten times higher than their chicks, and males had higher body weights and THg concentrations than females. The highest blood mercury concentrations (>2.5 $\mu g \cdot mL^{-1}$) were found on lakes with pH below 6.5. Adult feathers contained 7.65 to 21 $\mu g \cdot g^{-1}$ THg, and chick feathers contained 1.37 to 3.35 $\mu g \cdot g^{-1}$ THg.

Total mercury was examined in a number of other bird species. Bald eagle *(Haliaeetus leucocephalus)* eggs collected from 1990 to 1992 from the lower mainland of British Columbia and Vancouver Island were found to contain from 0.096 to 0.38 $\mu g \cdot g^{-1}$ THg (Elliott et al. 1996). Nine species of marine birds collected from 1978 to 1984 from the Quoddy region (Bay of Fundy) contained 0.037 to 7.05 $\mu g \cdot g^{-1}$ THg (Braune 1987b). The highest concentrations were found in the kidney and liver of double-crested cormorants *(Phalacrocoras auritus)*. The brain and muscle of this species had levels of <1.0 $\mu g \cdot g^{-1}$ THg, as did all tissues of most other species (Braune 1987b).

Relatively high overall THg levels were reported from a number of birds from the Great Whale area of northern Québec. Common mergansers contained an average of 17.5 $\mu g \cdot g^{-1}$ THg in liver tissue, and 1.3 $\mu g \cdot g^{-1}$ in muscle tissue. Herring gulls *(Larus argentatus)* contained 2.9 $\mu g \cdot g^{-1}$ in liver tissue, and 1.0 $\mu g \cdot g^{-1}$ in muscle tissue. Common mergansers from the Nottaway-Broadback-Rupert area (SE coast of James Bay) had elevated tissue concentrations similar to mergansers from Great Whale (Langlois et al. 1995).

Total mercury levels were significantly higher in feathers of adult and nestling ospreys from reservoirs than natural environments in northern Québec. Adult osprey feathers from reservoirs averaged 51.8 $mg \cdot kg^{-1}$ dw, and from natural lakes and rivers averaged 16.5 $mg \cdot kg^{-1}$ dw (DesGranges et al. 1998). Nestling feathers contained 37.4 and 7.0 $mg \cdot kg^{-1}$ dw, respectively, from these locations. Osprey eggs contained 0.2 ± 0.1 mg $THg \cdot kg^{-1}$ wet weight regardless of their location which was explained by the fact that eggs were laid prior to ice-out (therefore prior to high mercury exposure) on the water bodies under study in northern Québec.

A number of researchers have examined aquatic bird eggs in the vicinity of the Great Lakes. Concentrations in herring gull *(Larus argentatus)* eggs from several areas in the Great Lakes ranged from 0.13 to 0.88 $\mu g \cdot g^{-1}$ THg (Koster et al. 1996). The highest levels reported were for common tern *(Sterna hirundo)* eggs from Lake Ontario taken in 1971, of 1.1 $\mu g \cdot g^{-1}$ (Gilbertson 1974), and from Lake St. Clair in 1973, of 1.1 $\mu g \cdot g^{-1}$ (Stendell et al. 1976). Tree swallow *(Tachycineta bicolor)* eggs from Akwesasne (north-east of Cornwall), Long Point Tip

(Lake Ontario), Mud Creek (Lake Erie) and Wye Marsh (Georgian Bay) measured in 1991 had very low concentrations of 0.043 to 0.076 μg·g^{-1} (Bishop et al. 1995).

7.8 Non-biological media (App. B-8)

Most of the available information about MeHg in non-biological media in Canada has been published relatively recently because analytical techniques that permit detection of low levels were developed only in the late 1980s (Bloom 1989). Very few laboratories in the country are yet able to make MeHg measurements in water samples. Methylmercury measurements from Ontario and/or Québec have been reported for surface water, rain, snow, seston, sediment, peat porewater and flooded soils.

Information on THg in sediments in the Canadian environment is detailed in the report "Canadian Sediment Quality Guidelines for Mercury" (Environment Canada 1997), while the natural distribution of geological mercury as it relates to sediments in Canada is mapped and described in Friske and Coker (1995). For soil THg information, refer to Environment Canada (1996). Total mercury concentrations in non-biological media are not tabulated in the present report, except where they are supplemental to MeHg concentrations.

Precipitation in a remote area of north-western Ontario contained measurable concentrations of MeHg. Samples of rainwater taken in the early 1990s at the Experimental Lakes Area ranged from 0.017 to 0.049 ng·L^{-1} MeHg, or less than 1% of THg. In snow samples, MeHg concentrations ranged from 0.008 to 0.104 ng·L^{-1}, representing 0.8% to 2.9% of THg (St. Louis et al. 1995).

Water samples from a variety of surface waters at the Experimental Lakes Area had average MeHg concentrations ranging from 0.03 to 1.38 ng·L^{-1}. The higher concentration was from a pond after experimental flooding as part of an experimental reservoir project (Kelly et al. 1995). The lower concentration (0.03 ng·L^{-1}) was for water flowing from an upland area that lacked wetlands, while water from watersheds dominated by wetlands had average levels of 0.626 ± 0.126 ng·L^{-1} MeHg (St. Louis et al. 1994). Porewater from peat in wetlands contained 0.6 ng·L^{-1} MeHg, which was 10% of THg (Moore et al. 1995).

Water samples taken in the late 1970s from the mercury-contaminated Wabigoon/English River area near Dryden reportedly contained 0.22 to 1.3 ng·L^{-1} MeHg (Parks et al. 1989). While these values seem quite possible considering the high levels of mercury found in biota in the area, rigorous clean techniques for sampling and modern analytical methods were not developed at that time.

In the Ottawa and Gatineau Rivers, as much as 36% of THg in water samples was MeHg. Samples from the Ottawa River contained 2.3 ng·L^{-1} MeHg (26%), and from the Gatineau River, 4.1 ng·L^{-1} MeHg (36%). In the same study, water from Black Lake contained a lower concentration of MeHg (1.3 ng·L^{-1}), but the proportion of MeHg remained high, at 37% (Schintu et al. 1989).

In the Great Whale and Nottaway-Broadback-Rupert areas of northern Québec, water/seston samples taken in the summer of 1989-1991contained 0.45 and 0.94 ng·L^{-1} MeHg, respectively. Samples taken in the fall/winter from these areas contained 0.02 and 0.46 ng·L^{-1} MeHg, respectively. The MeHg levels were 9 to 31% of THg (Langlois et al. 1995). Methylmercury levels in water samples from natural lakes of northern Québec from 1994 to 1996 averaged 0.049 (± 0.004 SE) ng·L^{-1} ranging from 0.018 to 0.115 ng·L^{-1} (N=30; Lucotte et al. 1998).

Methylmercury levels were approximately 3% of THg.

In the flooded systems of the Robert Bourassa and Laforge-1 Reservoirs of northern Québec, MeHg levels in water samples averaged 0.28 ng·L^{-1} ranging from 0.03 to 0.85 ng·L^{-1} (N=87; 1994-1996; Thérien and Morrison 1998). Over the same period, MeHg in water from neighbouring natural lakes was lower averaging 0.05 ng·L^{-1} and ranging from 0.02 to 0.11 ng·L^{-1} (N=30). The percent of THg that was MeHg averaged 12% in the reservoirs and 3% in the lakes.

Flooded soils in the vicinity of the La Grande-2 Reservoir of northern Québec contained up to 0.043 µg·g^{-1} dw MeHg depending on the depth the soil sample was taken. Soils at 4-11 cm in depth had the highest concentration of 0.043 µg·g^{-1} MeHg (30%), while the deeper soils from 11-19 cm contained 0.0019 µg·g^{-1} MeHg (6%), the lowest amount. The surface soils from 0-4 cm contained an intermediate amount at 0.0085 µg·g^{-1} MeHg (6.6%; Mucci et al. 1995).

Surface sediments from a number of northern Québec lakes contained from 0.0001 to 0.0006 µg·g^{-1} dw MeHg (Mucci et al. 1995).

8.0 TOXICITY OF METHYLMERCURY TO PISCIVOROUS ORGANISMS

While this chapter is intended to describe the toxicity of MeHg to wildlife consumers of fish, because the mechanism of toxicity is similar to other mammals, and because of the abundance of assays using laboratory animals, numerous examples using laboratory animals are also described. Avian wildlife species exposed to excessive MeHg usually incur reproductive and behavioural deficits, while the mammalian piscivores most often suffer neurological effects.

The toxicity of a substance to individual organisms depends on the level of exposure. Assuming that piscivorous birds and mammals consume fish from the same lake, the estimates of daily consumption rates, the trophic level of the fish consumed, and the body weight of the animal all contribute significantly to methylmercury exposure when expressed on a per kg of body weight basis. According to the CCME (1998), the daily food consumption of the otter is approximately 10% of body weight and that of mink is 24%. Assuming that 100% of the otter's diet is from aquatic food sources; piscivorous fish are assumed to comprise roughly 20% of this, while the other 80% consists of lower trophic level fish. On the other hand, mink are assumed to eat exclusively lower trophic fish. As a result of this fact and the high percent of daily bodyweight consumed as fish, otters will have a higher MeHg contact rate than mink (US EPA 1997b). The same reasoning and calculations may be used for piscivorous birds, which with few exceptions (e.g., herons), eat only lower trophic level fish.

The protective effect of selenium against methylmercury toxicity to vertebrates has been known for many years (Ganther et al. 1972). Although efforts to determine the exact mechanism continue, selenium has been shown to bind mercury after demethylation of methylmercury in the liver. The compounds formed in this manner probably include both mercury-selenoproteins and HgSe (Cavalli and Cardellicchio 1995; Palmisano et al. 1995). Because selenium restores the activities of certain liver enzymes, and glutathione status involved in antioxidative defence mechanisms, there may be a biologically derived protection against MeHg toxicity (Hoffman and Heinz 1998).

The consistent finding of lower percent MeHg in bird and mammal livers may be because many vertebrate species possess a capability to detoxify and sequester mercury originating as methylmercury in the diet. The extent to which this capability is developed appears to be related to feeding habits and is most well developed in fish-eating marine mammals and the carnivorous polar bear (Dietz et al. 1990). A correlation between selenium and mercury have also been reported for several seabirds, although the Se-Hg ratio may be higher than the usual 1:1 (Elliott et al. 1992). The capacity and adaptability of this system to detoxify methylmercury is largely unknown. Variable detoxification among individuals of a single species (pilot whales) has been demonstrated; lactating females demonstrated a significantly diminished detoxifying capability (Caurant et al. 1996).

8.1 Mode of Toxicity

Methylmercury is a strong neurotoxicant for most vertebrates because they lack external barriers and substantial internal detoxifying systems. The central nervous system is the most sensitive tissue to MeHg exposure. Methylmercury is more toxic than other forms of mercury as it can pass through the blood-brain barrier and nuclear membranes to react directly with cellular components (Sloss 1995). As described above, the toxic effects of MeHg have been known to decrease with concurrent selenium exposure. The potential effect of selenium in reducing mercury toxicity has been reviewed by Cuvin-Aralar and Furness (1991).

With respect to MeHg uptake by animals, about 95% of an ingested dose of MeHg is absorbed into the bloodstream from the gastrointestinal tract, whereas inorganic Hg is less efficiently absorbed (7 to 15%; Wolfe et al. 1998). Methylmercury distributes to all parts of the body, readily crosses placental barriers, may enter the foetal brain, and is readily accumulated in growing animal fur, bird feathers or human hair. In humans, distribution to tissues may be complete in about two days, except for the brain, which takes about three days (Suzuki et al. 1991). The accumulation of mercury in the brain is greater in mammals than in fish (Zillioux et al. 1993).

Studies on the mechanism of MeHg passage across the blood-brain barrier have indicated that MeHg enters the capillary endothelial cell as a complex with the amino acid, cysteine. Attached to the thiol group of cysteine, MeHg enters the cell on the large neutral amino acid carrier. Given the presence of the MeHg-cysteine complex in plasma, it is thought that this may be the most common entry pathway into all mammalian cells (Suzuki et al. 1991). There even is some evidence that MeHg can cross the cell membranes of the blood-brain barrier by forming a compound that structurally mimics amino acids (Clarkson 1994). Researchers are not sure why brain cells are selectively damaged by MeHg.

Behavioural, physiological, histopathological, and biochemical demonstrations of toxicological effects of MeHg have been found for a variety of animals (Suzuki et al. 1991). Disruption of protein synthesis is one of the earliest biochemical effects (Clarkson 1994). An early symptom of MeHg poisoning in humans is paraesthesia (numbness and tingling) in the hands, feet and around the mouth (Sloss 1995), ataxia (loss of coordination), and gait or limb reflex dysfunction (Moore and Ramamoorthy 1984). Genotoxic (De Flora et al. 1994), developmental and reproductive effects (WHO 1989) have also been reported. Because MeHg can cross placental barriers, teratogenic and embyrotoxic effects have been found. A list of the clinical signs and symptoms demonstrated by victims of the "Minimata Bay disease" is found in Table 8.

Table 8. **Frequency of clinical signs and symptoms of the Minimata Bay disease where humans consumed fish and shellfish containing 10-35 $\mu g \cdot g^{-1}$ mercury for an extended period.**

Symptom or Sign	Frequency of Occurrence (%)
Constriction of visual fields	100
Sensory disturbance	100
Loss of coordination	94
Impairment of speech	88
Impairment of hearing	85
Impairment of gait	82
Tremor	76
Mental disturbance	71
Exaggerated tendon reflexes	38
Hypersalivation	24
Excessive sweating	24
Muscular rigidity	21
Chorea[a]	15
Pathologic reflexes	12
Athetosis[b]	9
Contractures[c]	9

Source: Chang 1979

Notes:

[a] Uncontrollable and irregular muscle movements, especially of the arms, legs, and face.

[b] Inability to maintain one's position of fingers and toes.

[c] An abnormal, often permanent shortening, as of muscle or scar tissue that results in distortion or deformity, especially of a joint of the body.

8.2 Toxicity to Mammals

The few existing toxicity studies on wildlife species [mink (*Mustela vison*) and otter (*Lutra canadensis*)] are supplemented with assays utilizing laboratory animals. Most of the studies on wildlife species give MeHg concentrations in food without reporting quantities fed per unit body weight. Estimates of methylmercury consumption on a body weight (bw) per day basis are made preferably by using food intake (FI) and body weight data provided in the original study. Otherwise average daily consumption for an average sized animal species may be used (Table 9). For captive female mink, the average FI:bw ratio is 0.16 kg food$\cdot$kg^{-1} bw per day (Bleavins and Aulerich 1981). The lowest-observed-adverse-effect-level (LOAEL) refers to lowest MeHg concentration tested at which ecologically relevant adverse effects are observed. The no-observable-adverse effect-level (NOAEL) refers to the highest concentration tested at which ecologically relevant adverse effects are not observed. Any tissue concentrations measured at the termination of experiments are reported in Appendix C-1.

8.2.1 Acute and Chronic Toxicity (Appendix C-1)

Wren et al. (1987a) found that a diet containing 1 mg$\cdot$kg^{-1} MeHg was lethal to female and male mink within 11 weeks. As such, mink were fed the treatment diet every other day for the remainder of the 6 month study (~15 weeks); no further mortalities or clinical effects occurred (C. Wren 1999, pers. com.). The researchers believed that these animals might have been additionally stressed by cold weather, causing higher sensitivity than might otherwise have been found. This conclusion is supported by experiments with two strains of mice that were more sensitive to MeHg toxicity at 8°C than at 22 or 38°C (Nomiyama et al. 1980). However, cold weather stress is realistic for the Canadian environment, making this study relevant. Higher mortality occurred among female than male mink presumably due to higher rates of food consumption per body weight for females (0.18 kg food·kg^{-1} bw per day) compared with males (0.10 kg food·kg^{-1} bw per day) for males (Wren et al. 1987a). The authors reported daily intakes of 180 and 100 ug MeHg·kg^{-1} bw per day for females and males, respectively. The reduced exposure (1 mg·kg^{-1} MeHg every other day) equates daily intake rates of 90 and 50 ug MeHg·kg^{-1} bw per day for females and males, respectively. The only sublethal effect reported once the exposure was reduced was that kits were born with a high mercury burden that decreased by five weeks of age. This was evidence of the importance of placental transfer but not transfer of MeHg via mother's milk (Wren et al. 1987a,b).

According to a recent study, fish from James Bay hydroelectric reservoirs may contain levels of MeHg that are toxic to mink. Three groups of female mink were fed daily with natural fish diets containing approximately 0.1, 0.5, and 0.9 mg MeHg·kg^{-1} (16, 80, 144 µg·kg^{-1} bw per day). The fish were both piscivorous and non-piscivorous species from the northern Québec reservoirs. The study had been originally designed to run for a two year period, however, over 50% of the mink in the 0.9 mg·kg^{-1} group died with signs of neurotoxicity between days 80 and 101 (11 to 14 weeks) of the study (Chamberland et al. 1996). This is the same length of exposure that lethal effects were noted for mink fed a similar dosage by Wren et al. (1987a) described above. No clinical signs, neurological damage or reproductive effects were found for the lower dosages (0.1, 0.5 mg MeHg·kg^{-1}; Laperle et al. 1998). Brown-coloured urine was observed after day 59 in some mink fed the intermediate and highest dosage, although the toxicological significance of this finding was unknown. The total urine protein and individual protein data corroborated the finding of Wobeser et al. (1976b; discussed below) of little or no renal damage at all dosage levels (Chamberland et al. 1996). The authors reported no confounding effects of other contaminants in the fish diet.

In a similar study during the 1970s, female mink were fed rations that were a mix of ranch mink feed and fish (freshwater drum from Lake Winnipeg), at concentrations of <0.1 mg·kg^{-1} (control), 0.25 mg·kg^{-1}, and 0.34 mg·kg^{-1} (<16, 40, 54 µg·kg^{-1} bw per day). After 145 days (21 weeks), MeHg was accumulated in the liver and kidney, but no clinical signs of MeHg toxicity or histopathological lesions were observed in any of the mink (Wobeser et al. 1976a).

Wobeser et al. (1976b) reported that female mink fed a diet containing 1.1 mg·kg^{-1} (176 µg·kg^{-1} bw per day) MeHg survived, although histopathological abnormalities including pale, yellow livers, lesions in the central nervous system, and axonal degeneration were evident. In addition, two of five mink showed reduced movement toward the end of the three-month (12 week) study, the researchers believed that more effects would have occurred had the exposure period been longer. Anorexia, ataxia and death resulted at the higher dosage of 1.8 mg MeHg·kg^{-1} (288 µg·kg^{-1} bw per day).

Female and male mink fed a very high concentration of 5 mg MeHg·kg^{-1} survived an average of one month (Aulerich et al. 1974). Using body weight and food consumption data provided in the original paper, this dietary dose is converted to 700 and 500 µg MeHg·kg^{-1} bw per day for female and male mink, respectively. The mink exhibited typical acute MeHg poisoning symptoms of ataxia, anorexia and limb paralysis prior to death. A diet containing 10 mg·kg^{-1}

inorganic Hg (mercuric chloride) for five months produced no effects. As these were the only concentrations administered for the two forms of mercury, LC_{50} and LOAEL values were not established.

The single acute toxicity study on otters, although somewhat limited in scope, suggested that otters are as sensitive as mink to MeHg poisoning in laboratory studies. An undescribed diet containing 2 $mg \cdot kg^{-1}$ (90 $\mu g \cdot kg^{-1}$ bw per day[18]) over 159 to 213 days (5.3 to 7.1 months) was lethal to two of three otters studied. In addition, at this dose, anorexia occurred in one, and ataxia in two of these otters (O'Connor and Nielsen 1981). The surviving otter displayed both toxic symptoms prior to its euthanasia at the end of the study. A diet of 4 $mg \cdot kg^{-1}$ MeHg (170 $\mu g \cdot kg^{-1}$ bw per day) caused anorexic and ataxic symptoms in all three otters over 101 to 123 days, after which the otters were euthanized. A diet containing 8 $mg \cdot kg^{-1}$ MeHg (370 $\mu g \cdot kg^{-1}$ bw per day) was lethal to one of three otters within 59 days, with toxic symptoms in all three otters prior to their euthanasia. Because of a high minimum treatment level, a chronic LOAEL and NOAEL (no-observed-adverse-effect-level) could not be determined. In the wild, however, otters may be more susceptible to MeHg poisoning than mink because approximately 20% of the otter diet may consist of higher trophic level fish and, therefore, higher MeHg concentrations (US EPA 1997a; Table 9).

No studies were found on sublethal effects to otters, although at least one study documented sublethal concentrations of mercury in captured otters based on the laboratory studies with mink and otter described above (Halbrook et al. 1994).

LABORATORY ANIMALS

A diet containing 0.25 $mg \cdot kg^{-1}$ MeHg was highly toxic to domestic cats (*Felis catus*) when administered 5 days per week over 90 days. The cats first showed convulsions at day 68, with a mean survival time of 78 days (Eaton et al. 1980). Methylmercury was less toxic to cats when fed as contaminated tuna fish, where 0.50 $mg \cdot kg^{-1}$ fed for up to 11 months caused biochemical changes (Chang et al. 1974), and 0.55 $mg \cdot kg^{-1}$ plus 0.29 $mg \cdot kg^{-1}$ Se for 188 days induced behavioural changes (Houpt et al. 1988). The latter two studies only indicated the MeHg concentration in food without describing the intake based on body weight.

Domestic cats fed a range of dosages of MeHg daily for up to 2 years showed neurological impairment and minor CNS pathology at the higher dosages. The cats were fed 0.06 mg kg^{-1} bw diet per day. The diet was a mix of contaminated and uncontaminated northern pike (Charbonneau et al. 1976). Doses were 0.05 (control), 0.14, 0.33, 0.76, 1.2, and 2.9 mg $THg \cdot kg^{-1}$ (3, 8.4, 19.8, 45.6, 73.8 and 176 μg $THg \cdot kg^{-1}$ bw per day). Periodical monitoring of the diet indicated that MeHg accounted for 95% of THg. Clinical signs of MeHg toxicity (ataxia, loss of balance and motor incoordination) occurred in groups receiving 176 $\mu g \cdot kg^{-1}$ bw per day after 14 weeks of treatment, and in groups receiving 74 $\mu g \cdot kg^{-1}$ bw per day after 40 weeks of treatment. Subtle neurological deficits (impaired "hopping reaction" and hypalgesia[19]) after 60 weeks of treatment occurred but did not intensify in groups receiving 46 $\mu g \cdot kg^{-1}$ bw per day (0.76 $mg \cdot kg^{-1}$). After two years, there were no overt clinical signs of toxicity at the two lowest dosages and the control (Charbonneau et al. 1976). These findings suggest that 20 $\mu g \cdot kg^{-1}$ bw per day is the approximate NOAEL and 46 $\mu g \cdot kg^{-1}$ bw per day is the LOAEL for chronic dietary exposure to methylmercury in domestic cats.

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[18] Daily intake rates were calculated by the original authors using measured body weights and food consumption data.
[19] Diminished sensitivity to pain.

Crab-eating macaques (*Macaca fascicularis*) exhibited insensitivity to touch and loss of tactile response (Rice 1989) when dosed with MeHg. Five monkeys were given 50 $\mu g \cdot kg^{-1}$ bw per day from birth to seven years. Clinical and neurologic examinations were carried out during the exposure period and for an additional six years. As an indicator of the latent effects of MeHg, objective neurological examinations performed at the end of the observation period revealed that the treated monkeys were clumsier and slower to react when placed in exercise cages than control monkeys (Rice 1989). Dosages of up to 50 $\mu g \cdot kg^{-1}$ bw per day fed in apple juice to macaques over 150 days caused no clinical symptoms and no significant difference in cholinesterase activity (Petruccioli and Turillazzi 1991).

Groups of Wistar rats (50 per sex per dose) were administered nominal daily doses of 2, 10, 25, 50 and 250 $\mu g \cdot kg^{-1}$ bw per day of MeHg for 26 months (Munro et al. 1980). Female rats that received 25 $\mu g \cdot kg^{-1}$ bw per day had reduced body weight gains and showed only minimal clinical signs of neurotoxicity; however, male rats that received this dose showed overt clinical signs of neurotoxicity, had decreased hemoglobin and hematocrit values, reduced weight gains, and showed increased mortality. Histopathological examination of rats of both sexes receiving 25 $\mu g \cdot kg^{-1}$ bw per day revealed demyelination[20] of dorsal nerve roots and peripheral nerves. Males had severe renal (kidney) damage, while females had minimal renal damage. This study reported a NOAEL of 5 $\mu g \cdot kg^{-1}$ bw per day and a LOAEL of 25 $\mu g \cdot kg^{-1}$ bw per day.

A two year feeding study of MeHg chloride was conducted on B6C3F1 mice (60 mice per sex per group) at doses of 0, 0.4, 2, and 10 $mg \cdot kg^{-1}$ (0, 40, 170, and 830 $\mu g \cdot kg^{-1}$ bw per day) to determine chronic toxicity and possible carcinogenic effects (Mitsumori et al. 1990). The mice were examined clinically during the study and neurotoxic signs characterised by posterior paralysis were observed in 33 males after 59 weeks and three females after 80 weeks in the 830 $\mu g \cdot kg^{-1}$ bw per day group. A marked increase in mortality and a significant decrease in body weight gain were also observed in the 830 $\mu g \cdot kg^{-1}$ bw per day male dose group, beginning at 60 weeks. Post mortem examination revealed toxic encephalopathy[21] consisting of neuronal necrosis of the brain and toxic peripheral sensory neuropathy[22] in both sexes of the high dose group. An increased incidence of chronic nephropathy[23] was observed in the 170 and 830 $\mu g \cdot kg^{-1}$ bw per day males. Based upon this study a NOAEL of 40 $\mu g \cdot kg^{-1}$ bw per day and a LOAEL of 170 $\mu g \cdot kg^{-1}$ bw per day was determined.

Mitsumori et al. (1983, 1984) administered diets containing 0, 0.4, 2 or 10 $mg \cdot kg^{-1}$ of methylmercuric chloride (0, 0.011, 0.05, and 0.28 $mg \cdot kg^{-1}$ bw per day in males; 0, 0.014, 0.064, and 0.34 $mg \cdot kg^{-1}$ bw per day in females) to 56 per sex per group Sprague-Dawley rats for up to 130 weeks. At week 104, survival was approximately 55, 45, 75, and 10% in control, low-, mid-and high-dose males, respectively, and 70, 75, 75 and 30% in control, low-, mid- and high-dose females, respectively. All males in the high-dose group had died by week 119. Body weight gain was significantly decreased in high-dose males and females after 44 weeks (approximately 10-20%).

Groups of 25 female and 25 male rats were administered methylmercuric chloride at dietary levels of 0, 0.1, 0.5 and 2.5 $mg \cdot kg^{-1}$ (0, 0.004, 0.02, and 0.1 $mg \cdot kg^{-1}$ bw per day) for two years. No significant effects were observed on growth or food intake except for a 6% decrease in body weight gain at 60 weeks in high-dose females. Survival was 72, 68, 48 and 48% in control, low-, mid- and high-dose males, respectively, and 76, 60, 64 and 56% in control, low-, mid- and high-dose females, respectively (statistical significance not reported). Increases in relative kidney weights were observed in both males and females at the highest dose (Verschuuren et al. 1976).

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[20] To destroy or remove the myelin sheath of (a nerve fiber), as through disease.
[21] Any of various diseases of the brain.
[22] A disease or an abnormality of the nervous system.
[23] A disease or an abnormality of the kidney (may be referred to as renal damage).

Chronic exposure to MeHg at levels too low to cause clinical signs of toxicity may cause deficiencies in the immune system of animals. The long-term bioaccumulation of MeHg may cause animals to be more susceptible to diseases that they might otherwise resist (Wolfe et al. 1998).

8.2.2 Reproductive and Developmental Toxicity (Appendix C-2)

Reproductive effects of MeHg in mammals include developmental effects that produce behavioural deficits in offspring, impaired fertility, and foetal death. Swimming ability, operant learning, avoidance, maze learning, and development of reflexes may be affected at low dosages. Effects that occur at successively higher dosages include changes in spontaneous activity, visual function, vocalisation, and convulsions (Chang and Annau 1984; Shimai and Satoh 1985; Eccles and Annau 1987). These effects occur because MeHg selectively targets the foetal brain. Females may also transfer MeHg to their young in milk, although this was previously dismissed as being unimportant in mink (Wren 1987). Foetal red blood cells may contain 30% more MeHg than maternal blood cells (c.f. Wolfe et al. 1998).

Gunderson et al. (1986) administered daily doses of 50-70 $\mu g \cdot kg^{-1}$ bw per day of MeHg to eleven crab-eating macaques throughout pregnancy which resulted in blood levels of 1080-1330 $\mu g \cdot L^{-1}$ in mothers and 1410-1840 $\mu g \cdot L^{-1}$ in offspring. When tested 35 days after birth the infants exhibited significant visual recognition deficits compared to control macaque offspring.

In another study, groups of seven or eight female crab-eating macaques were given 0, 50 or 90 $\mu g \cdot kg^{-1}$ bw per day of MeHg through four menstrual cycles (Burbacher et al. 1984). They were mated with untreated males and clinical observations were made for an additional four months. Two of seven 90 $\mu g \cdot kg^{-1}$ bw per day treated females aborted and three did not conceive during the four-month mating period; the other two females delivered live infants. Two of seven females of the 50 $\mu g \cdot kg^{-1}$ bw per day dose group aborted; the remaining females delivered live infants. All eight females of the control group conceived and six delivered live infants. These reproductive results were not statistically significant. Reproductive failure within dose groups could be predicted by blood Hg levels. The dams did not show clinical signs of MeHg poisoning during the breeding period or gestation but dosed with 90 $\mu g \cdot kg^{-1}$ bw per day for one year, four of seven showed adverse neurological signs.

Bornhausen et al. (1980) reported a decrease in operant behaviour performance in four-month-old Wistar rats whose dams had received from 5 to 50 $\mu g \cdot kg^{-1}$ bw per day of MeHg on days 6 through 9 of gestation. A statistically significant effect ($P<0.05$) was observed in offspring whose dams had received 10 and 50 $\mu g \cdot kg^{-1}$ bw per day during gestation. A LOAEL that could be consumed by pregnant female rats causing decreases in offspring operant behaviour of 10 $\mu g \cdot kg^{-1}$ bw was suggested.

8.2.3 Carcinogenic and Mutagenic Effects (Appendix C-3)

Very few studies implicate MeHg as a carcinogen. Based on inadequate data in humans and limited evidence of carcinogenicity in animals, the US EPA has classified MeHg as a *possible* human carcinogen (US EPA 1995b). Male mice exposed to methylmercuric chloride in the diet had an increased incidence of renal adenomas[24], adenocarcinomas[25], and carcinomas. Several negative cancer bioassays were also reported. Although genotoxicity test data suggest that MeHg is capable of producing chromosomal and nuclear damage, there are also abundant negative genotoxicity data (De Flora et al. 1994).

The results are contradictory as to whether MeHg is carcinogenic to rodents. Interpretation of two of the positive studies on mice was complicated by observation of tumours only at doses that exceeded the maximum tolerated dose (MTD)[26]. A fourth dietary study in mice and four separate dietary studies in rats failed to indicate carcinogenicity associated with MeHg exposure.

Methylmercuric chloride was administered in the diet of ICR mice at levels of 0, 0.4, 2 or 10 mg·kg^{-1} (0, 0.0375, 0.188, and 0.906 mg·kg^{-1} bw per day in males and 0, 0.0318, 0.114, and 0.785 mg·kg^{-1} bw per day in females). Sixty mice of each sex were fed the given dose level for 104 weeks (Hirano et al. 1986). Six mice per sex in each group were sacrificed at 26, 52 and 78 weeks. Complete histopathological examinations were performed on all animals found dead, killed *in extremis* or killed by design. Mortality, group mean body weights and food consumption were comparable to controls. The first renal tumour was observed at 58 weeks in a high-dose (0.906 mg·kg^{-1} bw) male, and the incidence of renal epithelial tumours (adenomas or adenocarcinomas) was significantly increased in high-dose males (1/32, 0/25, 0/29, and 13/26 in the control, low-, mid- and high-dose males, respectively). Focal hyperplasia of the tubular epithelium was reported to be increased in high-dose males (13/59; other incidences not reported). Epithelial degeneration of the renal proximal tubules and degeneration or fibrosis of the sciatic nerve was reported in high-dose females.

Mitsumori et al. (1981) administered 0, 15 or 30 mg·kg^{-1} of methylmercuric chloride in the diet (0, 1.6 and 3.1 mg·kg^{-1} bw per day) to 60 ICR mice per sex per group for 78 weeks. Interim sacrifices of up to 6 per sex per group were conducted at weeks 26 and 52. Survival was decreased in a dose-related manner; at week 78 survival was 40, 10 and 0% in control, low- and high-dose males, respectively, and 55, 30 and 0%, in control, low- and high-dose females, respectively (statistical analyses not performed). The majority of high-dose mice (85% males and 98% females) died by week 26 of the study. Examination of the kidneys of mice that died or were sacrificed after 53 weeks showed a significant increase in renal tumours in low-dose males (13/16 versus 1/37 in controls). Renal tumours were found in males but not in females of any group. Evidence of neurotoxicity and renal pathology were observed in the treated mice at both dose levels.

Mitsumori et al. (1983, 1984) administered diets containing 0, 0.4, 2 or 10 mg·kg^{-1} of methylmercuric chloride (0, 0.011, 0.05, and 0.28 mg·kg^{-1} bw per day in males; 0, 0.014, 0.064, and 0.34 mg·kg^{-1} bw per day in females) to 56 per sex per group of Sprague-Dawley rats for up to 130 weeks. No increase in tumour incidence was observed in either males or females. Intermediate dose (0.05 mg·kg^{-1} bw per day) males exhibited significantly increased degeneration of the proximal tubular epithelium and hyperplasia of the parathyroid.

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[24] A benign epithelial tumour having a glandular origin and structure.

[25] A malignant tumour originating in glandular tissue.

[26] The dose that an animal species can tolerate for a major portion of its lifetime without significant impairment or toxic effects other than carcinogenicity (US EPA website: www.epa.gov).

No increase in tumour incidence or decrease in tumour latency was observed in another study using rats of an unspecified species (Verschuuren et al. 1976); groups of 25 female and 25 male rats were administered methylmercuric chloride at dietary levels of 0, 0.1, 0.5 and 2.5 mg·kg^{-1} (0, 0.004, 0.02, and 0.1 mg·kg^{-1} bw per day) for two years. No effects on the nature or incidence of pathological lesions were observed, and tumours were reported to have been observed with comparable incidence and latency among all of the groups.

No increase in tumour incidence was observed in a multiple-generation reproduction study using Sprague-Dawley rats (Newberne et al. 1972). Groups of rats (30 per sex) were given semi-synthetic diets supplemented with either casein or a fish protein concentrate to yield dietary levels of 0.2 mg·kg^{-1} MeHg (0.008 mg·kg^{-1} bw per day). Another group of controls received untreated rat chow. Rats that received diets containing MeHg during the two-year study had body weights and haematology comparable to controls. Detailed histopathological analyses revealed no lesions of the brain, liver, or kidney that were attributable to MeHg exposure. Mortality data were not presented.

As reviewed in WHO (1990), MeHg is not a potent mutagen but appears to be capable of causing chromosome damage and nuclear perturbations under some circumstances. For the purposes of this report, MeHg will be considered non-mutagenic at levels found in Canadian environments, however, a few laboratory studies will be described.

MeHg produced chromosomal aberrations and aneuploidy[27] in human peripheral lymphocytes (Betti et al. 1992), sister chromatid exchange in human lymphocytes (Morimoto et al. 1982), and DNA damage in human nerve and lung cells as well as Chinese hamster V-79 cells and rat glioblastoma cells (Costa et al. 1991).

Bone marrow cells of cats treated with MeHg in a study by Charbonneau et al. (1976) were examined for mutagenic effects by Miller et al. (1979). The MeHg treatment caused an increased number of nuclear abnormalities and an inhibition of DNA repair capacity. Methylmercury induced a weak mutagenic response in Chinese hamster V-79 cells (Fiskesjo 1979). Methylmercury also induced histone protein perturbations and influenced factors regulating nucleolus-organising activity (WHO 1990). Moreover, MeHg has been reported to interfere with gene expression in cultures of glioma cells (WHO 1990).

Negative mutagenic findings have also been reported. Methylmercury acetate was reported to be negative in a mouse micronucleus assay (Heddle and Bruce 1977). Matsumoto and Spindle (1982) reported no significant increase in sister chromatid exchange in developing mouse embryos. They did report, however, that the developing mouse embryos were highly sensitive to *in vitro* treatment with MeHg.

8.3 Toxicity to Avian Species

As it does in mammals, MeHg readily penetrates the blood-brain barrier in birds and may cause brain lesions, spinal cord degeneration, and CNS dysfunctions. It is a potent neurotoxicant and embryo toxicant. Also similar to mammals, intestinal absorption of MeHg in birds is nearly 100%, while that for inorganic Hg is limited to a few percent (Scheuhammer 1987). The health concerns for the avian wildlife species are usually reproductive and behavioural deficits; mammalian quadrupeds most often suffer ataxia, anorexia and hindlimb paralysis.

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[27] Having a chromosome number that is not a multiple of the haploid number for the species.

Most of the studies on wildlife species give MeHg concentrations in food without reporting quantities fed per unit body weight. Estimates of methylmercury consumption on a body weight (bw) per day basis are made preferably by using food intake (FI) and body weight data provided in the original study. Otherwise average daily consumption for an average sized animal species may be used (Table 9). The lowest-observed-adverse-effect-level (LOAEL) refers to lowest MeHg concentration tested at which ecologically relevant adverse effects are observed. The no-observable-adverse effect-level (NOAEL) refers to the highest concentration tested at which ecologically relevant adverse effects are not observed. Any tissue concentrations measured at the termination of experiments are reported in Appendix C-4.

8.3.1 Acute and Chronic Toxicity

MeHg poisoning in birds may cause loss of appetite, leading to weight loss, progressive weakness in wings and legs, and difficulty flying, walking or standing (Scheuhammer 1987; Wolfe et al. 1998). Kidney disease and kidney lesions are associated with elevated dietary mercury (Nicholson and Osborn 1984; Snelgrove-Hobson et al. 1988; Rao et al. 1989). Brain residues are most diagnostic for acute mercury poisoning in birds (Wolfe et al. 1998), while blood concentrations have been related to chronic effects (Evers et al. 1998). Total mercury concentrations in liver and kidney tissues of piscivorous birds are insufficient criteria for making toxicologically relevant judgements of possible mercury toxicity because such a small proportion is MeHg, and inorganic Hg may be excreted from these tissues (Scheuhammer et al. 1998).

A recent study indicated contrasting protective effects of selenium on MeHg toxicity to adults compared with young mallards. Selenomethionine protected against MeHg poisoning in adult males, but it worsened the effects of MeHg on the hatching, survival, and growth of offspring, and produced more teratogenic effects (Heinz and Hoffman 1998). Except for one control that died of unknown causes, no adults that were fed a diet containing 10 mg·kg^{-1} MeHg plus 10 mg·kg^{-1} selenomethionine exhibited any overt signs of toxicity. The physiological protection of Se against MeHg poisoning was found to include restoration of plasma and liver GSH-peroxidase activity, partial restoration of liver and brain G-6-PDH activity, and lower hepatic oxidized glutathione (GSSG) concentration (Hoffman and Heinz 1998).

Mallards fed a diet containing 10 mg·kg^{-1} MeHg chloride showed overt signs of toxicity starting at about seven weeks. Males had weakened legs, and by day 73, one of 12 males died and eight others could barely walk (Heinz and Hoffman 1998). The researchers hypothesised that adult females did not suffer from MeHg poisoning during the study because they excreted the MeHg during egg laying, reducing their mercury body burdens (Heinz and Hoffman 1998).

8.3.2 Reproductive and Developmental Toxicity

The avian reproductive system is very sensitive to MeHg toxicity. Overall reproductive success in birds can decrease by 35 to 50% due to dietary MeHg exposures that would be insufficient to cause obvious signs of intoxication in adults (Wolfe et al. 1998).

In a field study of effects to loons (*Gavia immer*) over three years in the contaminated English-Wabigoon district of north-western Ontario, Barr (1986) reported differences in loon reproductive behaviour and success between contaminated and uncontaminated lakes. On Ball Lake, where mercury in perch (mean length 16.1 cm) averaged 0.36 mg·kg^{-1}, no nests were initiated even though loons were present on territories. Based on an adult bird size of 4 kg consuming 800 g of fish per day, this is equivalent to consuming 72 µg·kg^{-1} bw·d^{-1} (US EPA 1997b). In other areas of the contaminated section, Barr (1986) suggested that mercury levels in loon eggs may have been sufficiently high to reduce hatching success because they exceeded levels found to cause embryo mortality in other species. Reductions in egg laying, and in nest site and territorial fidelity were associated with mean mercury concentrations ranging from 0.3 to 0.4 mg·kg^{-1} (60 to 80 µg·kg^{-1} bw·d^{-1}) in perch and crayfish, and from 2 to 3 mg·kg^{-1} in adult loon brains and loon eggs. Loons established few territories, laid only one egg per pair, and raised no progeny in waters where mean contamination of small prey species exceeded 0.4 mg·kg^{-1} (Barr 1986).

Some male loons from mercury-contaminated Ball Lake were emaciated, which is a symptom of heavy metal poisoning (Borg et al. 1970). Because males consume more than females and eat larger fish, and do not excrete contaminants through egg laying, male loons were considered more susceptible to MeHg toxicity. Barr (1986) concluded that as mercury contamination increases, adult loons were more likely to fail to produce eggs, and that contaminated prey species caused aberrant behaviour in adult loons that resulted in reduced reproductive success. Lakes where no effects to loon behaviour and reproduction were observed contained perch with average mercury concentrations ranging from 0.08 to 0.17 mg·kg^{-1} (Barr 1986). The calculated NOAEL for 0.17 mg·kg^{-1} is 34 µg·kg^{-1} bw·d^{-1} based on the daily consumption of 800 g of food by a 4 kg loon (CCME 1998; US EPA 1997b).

The number of adult loons returning to their breeding territories in Wisconsin was found not to be influenced by differences in mercury exposure. Annual return rates of 85 to 90% were recorded for three years, indicating a high rate of adult survival despite blood mercury concentrations of up to 4.2 µg·mL^{-1}, and feather concentrations up to 21 µgg^{-1} ww (Evers et al. 1998). Fewer chicks hatched or survived to eight weeks of age on lakes where elevated mercury (>0.3 µg·mL^{-1}) was measured in chick blood. While measurements were made as THg, it is assumed that MeHg was ingested because the diet of these loons was fish (fish mercury concentrations were not reported).

Great blue herons (*Ardea herodias*) and double-crested cormorants appear to be more resistant to MeHg toxicity than loons. The reproductive success rates of herons and cormorants at a mercury-contaminated site in California were comparable to success rates from sites that were not contaminated (Wolfe and Norman 1998). The nesting colonies were on Clear Lake, which formerly had open-pit cinnabar mining and ore processing activities on its shoreline. Mercury waste continues to leach into the lake. The mercury concentration in food (minnows) recovered in and around nests, and the gastrointestinal tracts of heron young averaged 0.56 mg·kg^{-1} (range of 0.31-0.87 mg·kg^{-1}), and the mercury concentration in heron young brain tissue averaged 0.35 mg·kg^{-1}. Young cormorants had brain tissue concentrations of 0.63 (0.54 - 0.72 mg·kg^{-1}). The dietary intake for heron nestlings was calculated to be 73 to 210 µg MeHg·kg^{-1} bw·d^{-1} (using a food consumption rate of 0.245 kg·d^{-1} for a chick weighing 1.0 kg), with adults having an average rate of 122 µg MeHg·kg^{-1} bw·d^{-1} (CCME 1998). The number of eggs produced and the growth rates of young herons were no different than from colonies in uncontaminated sites from Washington and Canada (Wolfe and Norman 1998).

Feeding experiments with adult black ducks (*Anas rubripes*) caused a number of reproductive and developmental effects. Breeding pairs were fed 3 mg·kg^{-1} MeHg free-choice over two breeding seasons. Clutch size, egg production, number of eggs incubated, hatchability, and survival of ducklings were lower during both years in hens fed MeHg. Whole embryos that failed to hatch contained means of 9.6 and 6.1 mg·kg^{-1} mercury during the first and second years, respectively. Brains of dead ducklings were associated with lesions and contained from 3 to 7 mg·kg^{-1} mercury (Finley and Stendell 1978). By the second year of study, some adults displayed some behavioural effects characteristic of MeHg poisoning, but otherwise were healthy. Amounts of MeHg consumed was not reported either on a daily or total experimental basis.

Experiments that compared reproduction and behaviour over two reproductive seasons in mallard ducks fed diets contaminated with methylmercury indicated that effects were less severe in the second season than the first. Adult hens were fed through their first reproductive season diets containing nominal concentrations of 0 (control), 0.5 or 3.0 mg·kg^{-1} dw of MeHg; the measured concentrations were <0.05, 0.55, and 3.4 mg·kg^{-1} dw[28], respectively. Egg production ceased after 43, 31, and 21 weeks exposure to 0, 0.5, and 3.0 mg·kg^{-1} dw treatment diets, respectively. The only observed effect at the 0.5 mg·kg^{-1} dw treatment level within the first 21 weeks of exposure was that the eggs from treated females were smaller than controls (Heinz 1974). Those fed the more highly contaminated diet (3.0 mg·kg^{-1} dw) laid fewer eggs, had reduced hatching success and many of the young died within one week of hatching (results not quantified). By the second reproductive season, adult hens had been fed treatment diets continuously for 14 months. As in the first reproductive season, percent survival (89%) of young from hens fed 3.0 mg·kg^{-1} dw (measured as 2.88 mg·kg^{-1} dw [29]) was also lower than the survival of controls (98%), but the effect was less severe in the second season (Heinz 1976a). Ducklings from the treatment group that died had brain lesions characteristic of MeHg toxicity while those that survived were hyper-responsive in avoidance tests (Heinz 1975; 1976a). In contrast to the first reproductive season, egg production and hatching success for hens fed 3.0 mg·kg^{-1} dw were not significantly different from controls in the second reproductive season (Heinz 1976a). There were no consistent differences between the control and 0.5 mg·kg^{-1} dw (measured as 0.53 mg·kg^{-1} dw [30]) treatments with regards to proportion of eggs laid outside the nest, eggshell thickness, hatching success, or duckling survival to one week during the second reproductive season. With respect to behavioural responses, effects appeared also less severe in the second season than in the first. During the first season, offspring from both treatment groups were hyper-responsive in avoidance tests while during the second season only offspring from the 3.0 mg·kg^{-1} dw dose were hyper-responsive (Heinz 1975). Heinz (1976a) hypothesised that the combination of improved reproductive success and fewer behavioural effects for ducks in the 3.0 mg·kg^{-1} dw treatment group during the second reproductive season was due in part to a reduction in mercury levels in eggs and in part to a greater ability of adults and young to deal with methylmercury poisoning.

The adult mallards and their second season young in the above study served also as the first and second generations, respectively, of a three generation study (Heinz et al. 1976a;b, 1979). Beginning at 9 days of age, offspring were fed the same nominal dose as their mothers (0 or 0.5 mg·kg^{-1} dw; measured as 0.47 mg·kg^{-1} dw [31]). At adulthood, second generation treated mallards did not differ from controls in body weight or egg laying rates even though they consumed significantly more food. Treated hens displayed aberrant nesting behaviour and their offspring displayed reduced growth and survival, but not impaired behaviour (Heinz 1976b; Heinz 1979). These results were reversed in the third generation; third generation adults did not consume significantly more food and their offspring had growth rates comparable to controls.

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[28] For the first reproductive season, Heinz (1974) reports measured concentrations for the nominal dose 0.5 mg·kg^{-1} dw of 0.49, 0.53, and 0.64 mg·kg^{-1} dw; the mean of these three values is 0.55 mg·kg^{-1} dw. Measured concentrations for 3.0 mg·kg^{-1} dw were 3.32, 3.32, and 3.55 mg·kg^{-1} dw; the mean of these three values is 3.4 mg·kg^{-1} dw

[29] For the second reproductive season, the nominal dose of 3.0 mg·kg^{-1} dw was measured as 2.22, 2.83, and 3.59 mg·kg^{-1} dw; the mean is 2.88 mg·kg^{-1} dw (Heinz 1976a).

[30] For the second reproductive season, the nominal dose of 0.5 mg·kg^{-1} dw was measured as 0.52, 0.53, and 0.54 mg·kg^{-1} dw; the mean is 0.53 mg·kg^{-1} dw (Heinz 1976a). These concentrations apply also to the first generation of the three generation study.

[31] For the second and third generations, Heinz (1976b; 1979) reports the means of the measured concentrations as 0.47 and 0.43 mg·kg^{-1} dw, respectively, for the nominal dose of 0.5 mg·kg^{-1} dw. Results from second and third generation studies were not reported for a 3.0 mg·kg^{-1} dw treatment level as in the earlier publications.

Third generation offspring, however, displayed impaired behaviour; fewer ducklings took more time to approach a tape-recorded maternal call (Heinz 1979).

The effect of MeHg on mallard development was studied by applying quantities on eggs externally. Eggs were treated on day 3 of development with microlitre applications of MeHg in a carrier. Dose-related effects on survival, growth and abnormal development were recorded. Almost half of the mercury applied entered the eggs past the shell membranes within several days of treatment. Malformations occurred at 1 μg·egg^{-1} (18 μg·kg^{-1}) [32]; decreased embryonic growth at 9 μg·egg^{-1} (164 μg·kg^{-1}). With the application of 27 μg·egg^{-1} (490 μg·kg^{-1}), only 63% of the embryos were alive by 18 days of development (Hoffman and Moore 1979). Residue analyses were made that could be compared with egg concentrations in field studies.

8.4 Toxicity to Piscivorous Fish

Fish appear to be tolerant to large body burdens of MeHg. Fingerling rainbow trout that were fed a diet containing 4, 8, 16 and 24 mg·kg^{-1} methylmercuric chloride survived and exhibited few signs of toxicity even at the highest dosages (Wobeser 1975). Body concentrations were consistently higher than the food concentrations, with over 30 mg·kg^{-1} measured in a fish fed the 24 mg·kg^{-1} dosage for 15 weeks. In the final five weeks of the experiment, fish fed the two highest dosages showed significantly lower weight gains than the lower dosage groups. The lack of damage to the nervous system was thought to be because the MeHg did not reach toxic levels.

Realistic levels of MeHg in the diet caused sublethal effects to juvenile walleye. Friedmann et al. (1996) fed groups of walleye either 0, 0.1 or 1.0 μgg^{-1} MeHg that had been injected into catfish or fathead minnow tissue. After six months, the higher dosage significantly impaired fish growth (length, weight) as well as gonadal development in the male but not the female fish. Pathohistological analysis indicated that testes of fish fed the high (1.0 μgg^{-1}) MeHg diet showed significant alterations with multifocal cell atrophy compared to controls. Some testicular atrophy was observed to a lesser degree in fish fed the low (0.1 μgg^{-1}) MeHg diet. Ovaries from both control and MeHg fed fish did not contain any predominant lesions. Adult walleye in the wild are more likely to consume lower trophic level fish, which realistically, may contain the lower concentration of MeHg tested here.

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[32] Based on an average mallard egg weight of 55 g (Wolfe et al. 1998).

Table 9. Consumption parameters for mink, otter, kingfisher, loon, osprey, and eagle

Species	Body Weight (kg)	Ingestion Rate or Food Intake ($kg \cdot d^{-1}$)	Drinking Rate $L \cdot d^{-1}$	Trophic Level of Food Source	% diet at each trophic level	Reference
Mink Wild Wild female Captive male Captive female	0.80 0.6 1.82 0.87	0.178 0.143 0.22 0.14	0.081	3	90	US EPA 1997b CCME1998 Bleavins and Aulerich 1981
Otter	7.40 8.0	1.220 0.8	0.600	3,4	80,20	US EPA 1997b CCME 1998
Belted Kingfisher	0.15	0.075	0.017	3	100	US EPA 1997b
Loon	4.0 4.134	0.8 0.73	0.14	3	100	US EPA 1997b CCME 1998
Osprey	1.50	0.300	0.077	3	100	US EPA 1997b
Eagle	4.60 4.5	0.50 0.5	0.160	3,4,5[a]	74,18,8	US EPA 1997b CCME 1998

Notes:

[a] Level 5 indicates the eagle may consume other piscivorous birds.

9.0 EXISTING TISSUE RESIDUE GUIDELINES

9.1 Human Health

There are two basic methods of setting human consumption guidelines. In the past, the levels were usually based on a concentration in tissue to be consumed. This is still a practical way for jurisdictions to know how much mercury is in fish or shellfish, but the levels are not necessarily related to the amount of mercury consumed by an individual. Increasingly, guidelines are based on a daily or weekly dosage (i.e., $\mu g\ MeHg \cdot kg^{-1}\ bw \cdot d^{-1}$) considered to be safe for consumption.

The Canadian and the WHO guidelines for food safety, based on technical reports from annual meetings of the joint FAO/WHO Expert Committee on Food Additives, recommend that the total concentration of mercury in the edible portion of commercial fish must not exceed $0.5\ \mu g \cdot g^{-1}$ on a wet weight basis (WHO 1972, 1976, 1990, HWC 1990; OECD 1994). Swordfish and shark are exempted, and consumption of these species should not exceed one meal per week. Many other countries have adopted the FAO/WHO tissue guideline for commercial fish consumption of $0.5\ \mu g \cdot g^{-1}$, and relatively few countries have set the higher limit recommended by the US EPA, of $1.0\ \mu g \cdot g^{-1}$ (see Table 10).

Canada has qualified the concentration-based value on the frequency of consumption, which effectively considers the dosage of mercury consumed. Women of childbearing age and children should consume only fish at or below $0.5\ \mu g \cdot g^{-1}$. From 0.5 to $1.0\ \mu g \cdot g^{-1}$, the average adult eating fish temporarily over a short period may consume up to ten meals per week (2300 g). If consumed throughout the year, fish containing 0.5 to $1.0\ \mu g \cdot g^{-1}$ MeHg should be consumed only once per week (200 g). Above $1.0\ \mu g \cdot g^{-1}$, the average adult eating fish temporarily over a short period should consume no more than seven meals per week (1500 g), and if eaten throughout the year, fish measuring above $1.0\ \mu g \cdot g^{-1}$ should be consumed no more than once per week (portion of 140 g). In Canada, fish measuring over $1.5\ \mu g\ MeHg \cdot g^{-1}$ are not recommended for human consumption at any time (Appendix D; WHO 1972; 1976; HWC 1990).

The WHO evaluated relationships between response and body burden (hair or blood mercury concentrations) and determined that no adverse effects (NOAEL) were detected with long-term daily methylmercury intakes of 3-7 $\mu g \cdot kg^{-1}$ bw (hair mercury concentrations of approximately 50-125 $\mu g \cdot g^{-1}$). They recommend that adults consume no more than 0.23 mg MeHg per week, or $0.47\ \mu g \cdot kg^{-1}\ bw \cdot d^{-1}$ (Table 10). They postulate that pregnant women (i.e., foetus) may suffer effects at lower methylmercury exposure levels than non-pregnant adults, suggesting a greater risk for pregnant women (WHO 1990).

The U.S. Food and Drug Administration (FDA) advises persons other than pregnant women and women of child-bearing age to limit their consumption of fish species with methylmercury levels of $1\ \mu g \cdot g^{-1}$ to approximately 200 g per week (one serving). For fish with levels averaging $0.5\ \mu g \cdot g^{-1}$, regular consumption should be limited to about 500 g per week (two servings).

The FDA action level is based on consideration of the tolerable daily intake (TDI) for MeHg, as well as information on seafood consumption and associated exposure to MeHg. The TDI is the amount of MeHg that can be consumed daily over a long period of time with a reasonable certainty of no harm to adults. The U.S. FDA (and WHO) established a TDI based on a weekly tolerance of 0.3 mg of THg per 70 kg person, of which no more than 0.23 mg should be present as MeHg. These amounts are equivalent to 5 and $3.3\ \mu g \cdot kg^{-1}$ bw, respectively. Using the values for MeHg, this tolerable level would correspond to approximately 230 µg per week for a 70 kg person or 33 µg per person per day, which is the same level of intake recommended for long term, frequent consumption of fish in Canada. The TDI was calculated from data developed in part by Swedish studies of Japanese individuals poisoned in the episode of Niigata which resulted from the consumption of contaminated fish and shellfish and the consideration of other studies of fish-eating populations (US EPA 1997a).

The US EPA has designated a reference dose (RfD) for humans of 0.1 µg MeHg·kg^{-1} bw·d^{-1}. Another agency, the ATSDR (Agency for Toxic Substances and Disease Registry) formerly recommended a minimum risk level (MRL) of 0.1 µg·kg^{-1} bw·d^{-1}, but they have increased this value to 0.3 µg·kg^{-1} bw·d^{-1} based on the results of new studies of fish and marine mammal consuming populations in the Seychelles Islands (Indian Ocean) and Faroe Islands (North Atlantic Ocean) (US EPA 1997c; ATSDR 1999)[33]. Some are concerned that such a relaxation of the ATSDR standard could jeopardise efforts to curb mercury emissions (Renner 1998). Note that the ATSDR value is a standard (enforceable by law), whereas the EPA RfD is a guideline only.

Thus far, jurisdictions have emphasised reducing the consumption of contaminated fish rather than reducing mercury inputs that cause fish levels to be elevated. This has implications to aboriginal populations that derive important nutritional benefits through consumption of fish. According to the Cree Board of Health and Social Services of James Bay, in most instances limiting fish consumption to prevent mercury contamination carries risks that may be higher than the mercury itself (C. Dumont 1999, pers. com.). They note that fish consumption helps to prevent diseases such as heart disease, diabetes, mellitus and arthritis.

9.2 Wildlife

The US EPA has set water quality guidelines that are associated with tissue residue guidelines (reference doses, RfD) to protect mammal and bird consumers of fish. A wildlife criterion (WC) value is defined as the concentration of mercury in water which, if not exceeded, protects avian and mammalian wildlife populations from adverse effects resulting from ingestion of surface waters and from ingestion of aquatic life taken from these surface waters. Thus, the WC is the highest aqueous concentration (dissolved and colloid bound) of mercury that causes no significant reduction in growth, reproduction, viability or usefulness (in a commercial or recreational sense) of a population of animals exposed over multiple generations (US EPA 1997b).

In the U.S., two separate criteria values, expressed as filtered water concentrations, were derived for THg and MeHg. The total Hg criteria was based on a model proposed by the GLWQI (Great Lakes Water Quality Initiative; US EPA 1995c,d), and the MeHg values (WC) were developed as a modification of the GLWQI model by US EPA in their "Mercury Study Report to Congress" (US EPA 1997b,c). The models are based on potential bioaccumulation (BAF[34]) from water and from diet of the mercury species. Because the use of THg rather than MeHg was a source of criticism of the initially proposed WC (and BAF) values, US EPA developed new BAF results for MeHg. The GLWQI was developed based on a NOAEL for piscivorous wildlife of 1.1 ppm, compared with a NOAEL in the report to Congress of 0.33 ppm (both from Wobeser's research, 1975, 1976). The GLWQI value employed an uncertainty factor (UF) of ten, compared with an UF of three in the report to Congress.

The GLWQI for THg was intended to protect wildlife in the Great Lakes region with a value of

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[33] April 19, 1999 update: http://www.atsdr.cdc.gov
[34] BAF = bioaccumulation factor

1300 pg·L^{-1} proposed (US EPA 1997b). The US EPA report to Congress recommended a WC for protection of piscivorous wildlife species of 50 pg MeHg·L^{-1}, which was the lower value of the calculated average avian wildlife criterion of 61 pg·L^{-1} and average piscivorous mammalian wildlife criterion of 50 pg·L^{-1}. This value corresponds to a *total* mercury concentration in the water column of 641 pg·L^{-1} [35] and MeHg concentrations in fish of 0.077 ppm (forage fish = trophic level 3) and 0.346 ppm (piscivorous fish = trophic level 4). The WC, like the human RfD, is predicted to expose wildlife to a safe dose over a lifetime. Recommended water concentrations of 50 pg MeHg·L^{-1} and 641 pg THg·L^{-1} are intended to protect avian wildlife from ingesting more than the safe daily dose i.e., the RfD of 21 µg·kg^{-1} bw·d^{-1}, and to protect semi-aquatic mammals from ingesting more than 18 µg·kg^{-1} bw·d^{-1}(US EPA 1997a,b).

The US EPA criteria for wildlife differs fundamentally from Canadian guidelines in that they are based on water concentrations rather than on tissue concentrations that might be consumed by piscivorous wildlife. In other words, the US EPA values originate several steps below the trophic level that the wildlife are feeding on. Given the complexity of mercury behaviour in the environment, including the fact that there may be no relationship between mercury in the water column (especially total Hg), and mercury found in fish, a criterion based on BAF values calculated from water concentrations is less likely to accurately predict MeHg in animal tissues. The report to Congress emphasises that there is a high degree of uncertainty with the BAF calculations. However, at least one study was supportive of the GLWQI (THg) value on a contaminated lake in California (Wolfe and Norman 1998). Also, US EPA's report to Congress did consider a daily dose in the diet (RfD) that should result in no adverse effects.

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[35] THg water concentration assumes a U.S. national average of 7.8% MeHg.

Table 10. Worldwide survey of mercury tissue residue guidelines (concentrations or daily limits) for mercury in fish and fisheries products.

Country or Agency	Human Consumption Guideline	Wildlife TRG	Reference
Australia	0.5 $\mu g \cdot g^{-1}$ in fish, crustaceans, molluscs	–	OECD 1994
Brazil	0.5 $\mu g \cdot g^{-1}$ - legal limit for hazardous metals	–	US EPA 1989, c.f. Haines et al. 1994
Canada	0.5 $\mu g \cdot g^{-1}$ in fish[a] 0.2 $\mu g \cdot g^{-1}$ for subsistence fish consumers[b]	0.033 $\mu g \cdot g^{-1}$ (this report)	HWC 1990; WHO 1972, 1976
Denmark	0.5 $\mu g \cdot g^{-1}$ - legal limit for hazardous metals	–	US EPA 1989, c.f. Haines et al. 1994
Equador	1 $\mu g \cdot g^{-1}$ - legal limit for hazardous metals	–	US EPA 1989, c.f. Haines et al. 1994
FAO/WHO	0.5 $\mu g \cdot g^{-1}$ in fish 0.23 mg MeHg per week (70 kg person) 0.47 μg MeHg$\cdot kg^{-1}$ bw$\cdot d^{-1}$	–	WHO 1972 WHO 1990 & Codex Alimentarius 1998
Finland	0.5-1.0 $\mu g \cdot g^{-1}$, once per week >1.0 $\mu g \cdot g^{-1}$, no consumption	–	OECD 1994
France	0.5-0.7 $\mu g \cdot g^{-1}$ - legal limit for hazardous metals	–	US EPA 1989, c.f. Haines et al. 1994
Germany	1 $\mu g \cdot g^{-1}$ - legal limit for hazardous metals	–	US EPA 1989, c.f. Haines et al. 1994
Great Britain	0.2 mg MeHg per week (FAO/WHO)	–	OECD 1994
Greece	0.7 $\mu g \cdot g^{-1}$ – legal limit for hazardous metals	–	US EPA 1989, c.f. Haines et al. 1994
Hong Kong	0.5 $\mu g \cdot g^{-1}$ – legal limit for hazardous metals	–	US EPA 1989, c.f. Haines et al. 1994
India	0.5 $\mu g \cdot g^{-1}$ – legal limit for hazardous metals	–	US EPA 1989, c.f. Haines et al. 1994
Israel	0.5 $\mu g \cdot g^{-1}$ – legal limit for hazardous metals	–	US EPA 1989, c.f. Haines et al. 1994
Italy	0.7 $\mu g \cdot g^{-1}$ – legal limit for hazardous metals	–	US EPA 1989, c.f. Haines et al. 1994
Japan	0.3-0.4 $\mu g \cdot g^{-1}$ - legal limit for hazardous metals	–	US EPA 1989, c.f. Haines et al. 1994
Korea	0.5 $\mu g \cdot g^{-1}$ – legal limit for hazardous metals	–	US EPA 1989, c.f. Haines et al. 1994
Netherlands	1 $\mu g \cdot g^{-1}$ limit for hazardous metals in fish 0.5 $\mu g \cdot kg^{-1}$ bw$\cdot d^{-1}$ adults 0.1 $\mu g \cdot kg^{-1}$ bw$\cdot d^{-1}$ pregnant/lactating women	–	US EPA 1989, c.f. Haines et al. 1994 OECD 1994
New Zealand	0.5 $\mu g \cdot g^{-1}$ - legal limit for hazardous metals	–	US EPA 1989, c.f. Haines et al. 1994
Philippines	0.5 $\mu g \cdot g^{-1}$ - legal limit for hazardous metals	–	US EPA 1989, c.f. Haines et al. 1994
Spain	0.5 $\mu g \cdot g^{-1}$ - legal limit for hazardous metals	–	US EPA 1989, c.f. Haines et al. 1994
Sweden	1 $\mu g \cdot g^{-1}$ - legal limit for hazardous metals	–	US EPA 1989, c.f. Haines et al. 1994

Country or Agency	Human Consumption Guideline	Wildlife TRG	Reference
Switzerland	0.5 $\mu g \cdot g^{-1}$ - legal limit for hazardous metals	–	US EPA 1989, c.f. Haines et al. 1994
Thailand	0.5 $\mu g \cdot g^{-1}$ - legal limit for hazardous metals	–	US EPA 1989, c.f. Haines et al. 1994
US ATSDR[c]	0.5 $\mu g \cdot kg^{-1}$ $bw \cdot d^{-1}$, Nov. 1997 increased from 0.1 $\mu g \cdot kg^{-1}$ $bw \cdot d^{-1}$	–	US EPA 1997c
US EPA	0.1 $\mu g \cdot kg^{-1}$ $bw \cdot d^{-1}$	21 $\mu g \cdot kg^{-1}$ bw per d birds 18 $\mu g \cdot kg^{-1}$ bw per d mammals[d]	US EPA 1997c
US Food & Drug Admin.	1.0 $\mu g \cdot g^{-1}$	–	US EPA 1997c
Venezuela	0.1-0.5 $\mu g \cdot g^{-1}$ - legal limit for hazardous metals	–	US EPA 1989, c.f. Haines et al. 1994
World Health Organization - see FAO/WHO			
Zambia	0.2-0.3 $\mu g \cdot g^{-1}$ - legal limit for hazardous metals	–	US EPA 1989, c.f. Haines et al. 1994[e]

Notes:

[a] WHO/Canada- Shark, tuna, and swordfish exempt should be eaten no more than once per week.

[b] Subsistence level issued by Health Canada's Medical Services Branch in 1979, and reiterated in 1984 (N.P. McEwen 1999, Health Canada, pers. com.; App. D)

[c] Agency for Toxic Substances and Disease Registry

[d] US EPA wildlife TRG (RfD) are in review, and are based on wildlife criteria (WC) which are water concentrations of 50 pg $MeHg \cdot L^{-1}$ and 641 pg total.

[e] Haines et al. 1994 comprises the supporting documentation for MacDonald 1994.

10.0 CANADIAN TISSUE RESIDUE GUIDELINES FOR METHYLMERCURY

10.1 Protocol

TRGs are concentrations of environmental contaminants (that have a potential to biomagnify in the tissues of aquatic organisms; e.g., fish) recommended to protect wildlife that consume aquatic biota in freshwater, estuarine, or marine ecosystems. The protocol for the derivation of Canadian tissue residue guidelines (TRGs) for the protection of wildlife that consume aquatic biota is fully described in "Protocol for the derivation of Canadian tissue residue guidelines for the protection for wildlife that consume aquatic biota" (CCME 1998). While the protocol promotes a standard method of derivation, it is intended as a flexible procedural guide that should not replace best scientific judgement when developing guidelines. Note that the current protocol is intended as a working document for the testing and application of the methodology (CCME 1998).

Briefly, TRG derivation follows the evaluation of toxicological data from a range of avian and mammalian species. The goal is to protect the most sensitive species and life stages of wildlife. Certain minimum toxicological data set requirements must be met (see 3.3.1 - 3.4.3 in CCME 1998). The steps in derivation include:

1. Selecting high quality chronic studies from which LOAEL and/or NOAEL values are available; values may be adjusted to daily intake rates in units of mg·kg^{-1} bw per day if not originally presented as such

2. Selecting an Uncertainty Factor (UF) based on the reality of accurate extrapolation from differences in gender, life stage, other species, lab-to-field, and other factors;

3. Calculation of Tolerable Daily Intakes (TDI) in mg·kg^{-1} bw per day as the geometric mean of the selected LOAEL and NOAEL[36] divided by the UF. TDIs are calculated separately for mammalian and avian wildlife species.

4. Calculation of Reference Concentrations (RCs) for a wide range of wildlife species, where RC = TDI X (Body Weight/Food Intake); bw=kg; FI=kg·d^{-1}).

As the lowest TDI will not necessarily result in the lowest acceptable dietary concentration, the avian and mammalian TDIs are used in conjunction with the body weights (bw) and daily food intake rates (FI) of Canadian wildlife species to calculate RCs. The lowest RC is carried forward as the TRG. For the purposes of deriving national TRG values, the mammalian and avian RCs must be as inclusive as possible to accommodate all species and regions in Canada. Therefore, the derivation is based on the highest mammalian and avian FI:bw ratios known for Canadian wildlife, namely 0.24 for mink and 0.94 for Wilson's storm petrel (CCME 1998).

10.2 Guideline Derivation and Rationale

As detailed in chapter 8, there are a number of studies from both laboratory and wildlife species on MeHg toxicity. According to the CCME protocol, wildlife species are preferred and as such, high quality studies on mallards and minks were considered in calculating the TRG.

10.2.1 Avian Species

Three generations of mallard ducks fed a contaminated diet indicated that successive generations are susceptible to MeHg effects although there was no evidence that the effects became progressively more severe from one generation to the next (Heinz 1979). A dietary concentration of 0.5 mg·kg^{-1} dw of methylmercury is the LOAEL because it resulted in reduced growth and survival in offspring of second generation hens and in ecologically-relevant behavioural effects in offspring of third-generation hens (Heinz 1976b, 1979). In addition, hens fed 0.5 mg·kg^{-1} dw laid a greater percentage of eggs outside their nest boxes than control birds although the effect was statistically significant only in the second generation and when results for all three generations were combined (Heinz 1979). The nominal dietary concentration of 0.5 mg·kg^{-1} dw is equivalent to an intake rate of 75 µg·kg^{-1} bw per day assuming a measured dietary concentration of 0.48 mg·kg^{-1} dw[37] and a food consumption rate of 0.156 kg of dry food per kg of body weight per day[38] (Heinz 1979). The NOAEL of 13 µg·kg^{-1} be per day was calculated by dividing the LOAEL by 5.6 (CCME 1993). This calculated NOAEL of 13 µg·kg^{-1} bw per day is higher than the intake rate (6.4 µg·kg^{-1} bw per day) calculated for those birds fed control feed (0.05 mg·kg^{-1} dw; 0.128 kg of dry food per kg of body weight per day).

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[36] NOAEL may be estimated if necessary as LOAEL ÷ 5.6 (CCME 1993)

[37] The measured dietary concentration is 0.48 mg·kg^{-1} dw, when the means for each generation (0.53, 0.47, and 0.43 mg·kg^{-1} dw) are averaged (Heinz 1979). Individual measured concentrations could not be used because they were not reported for the second and third generations Heinz 1976b; 1979).

[38] When results were combined, second and third generation mallards fed the 0.5 mg·kg^{-1} dw treatment diet consumed 0.156 kg of dry food per kg of body weight per day; food consumption of the first generation was not recorded (Heinz 1979).

The avian TDI of 31 µg·kg^{-1} bw per day (Table 11) was calculated as the geometric mean of the LOAEL (75 µg·kg^{-1} bw per day) and NOAEL (13 µg·kg^{-1} bw per day) without the application of an uncertainty factor (UF). Not applying an UF is justified in this case because the experiments were conducted on a Canadian wildlife species (mallard), over three generations (a true chronic study), and at MeHg dosages that would commonly be found in wild fish. Dividing the TDI by the highest FI:bw ratio for wild birds (0.94 for Wilson's storm petrel) results in an avian RC of 33µg·kg^{-1} diet on a wet weight basis (Tables 11 and 12).

For perspective, the avian TDI of 31 µg·kg^{-1} bw per day is comparable to the reference dose (RfD) recommended by the US EPA (21 µg·kg^{-1} bw per day) to protect avian wildlife (US EPA 1997a), although the derivation procedures of the two values are not the same. The avian TDI is about 2.4 times lower than the daily ingested dose that resulted in the LOAEL for mallards discussed above. In contrast, the avian TDI is 66 times higher than the daily intake recommended by the WHO for humans (0.47 µg·MeHg·kg^{-1} bw per day; WHO 1990).

Weaknesses in the critical study used to calculate the avian RC include the fact that the mercury dosage was added to dry food (rather than using contaminated fish), and was fed to a species (mallard) that is not principally a piscivore. Moreover, although a dietary concentration of 0.5 mg·kg^{-1} dw of methylmercury produced adverse effects in all three generations, effects were not consistent from one generation to the next. In addition, because effects were found at the lowest test dosage, the NOAEL had to be calculated from the LOAEL. Nevertheless, the long-term chronic toxicity experiment completed by Heinz was the most comprehensive MeHg study available.

10.2.2 Mammalian Species

A study on domestic cats indicated that cats are sensitive to MeHg exposure (Charbonneau et al. 1976). As wild cats rarely consume aquatic biota, and because good quality studies on mink were available, the cat and Wistar rat assays (Munro et al. 1980) were not used for the derivation, but were referred to for comparison purposes only.

The most sensitive results from wildlife-based studies included reduced fertility and neurotoxic effects in female mink followed by death (>50%) at 0.9 mg·kg^{-1} (144 µg·kg^{-1} bw per day) (Chamberland et al. 1996). No ecologically relevant effects including clinical signs, neurological damage or impaired reproduction were observed in female mink at 0.5 mg·kg^{-1} (80 µg·kg^{-1} bw per day) and as such, this exposure was used as the NOAEL (Chamberland et al. 1996; Laperle et al. 1998).

Strengths of the critical study include the use of contaminated fish from James Bay reservoirs, a critical wildlife species (mink), and three treatment levels (Chamberland et al. 1996). Neurological effects considered ecologically relevant include ataxia, staggering, splaying or paralysis in the hind limbs, and anorexia. These effects and others were reported in many of the studies that described MeHg neurotoxicity. Such effects would render an animal unable to properly feed, escape from predators, reproduce, and would increase its susceptibility to other stressors.

The TDI for mammals of 22 µg·kg^{-1} bw per day was calculated as the geometric mean of the LOAEL (144 µg·kg^{-1} bw per day) and NOAEL (80 µg·kg^{-1} bw per day) divided by an uncertainty factor of 5. An UF of 5 was chosen because the endpoint of the LOAEL was death, the study lasted just over three months (subchronic), and to permit extrapolation to other wildlife species. A mammalian RC of 92 $\mu g \cdot kg^{-1}$ diet on a wet weight basis was obtained by dividing the TDI by the highest FI:bw ratio for wild mammals (0.24 for female mink; Tables 8 and 9).

The mammalian TDI is comparable to the US EPA's reference dose (RfD) for semi-aquatic mammals of 18 $\mu g \cdot kg^{-1}$ bw per day and is approximately equal to the chronic NOAEL (20 $\mu g \cdot kg^{-1}$ bw per day) estimated from a two-year feeding study with domestic cats (Charbonneau et al. 1976). The mammalian TDI is 47 times higher than the intake recommended by the WHO for humans (0.47 $\mu g \cdot kg^{-1}$ bw per day; WHO 1990).

10.3 Methylmercury Guideline

For the protection of all life stages of all birds and mammals that consume aquatic organisms, the lowest RC, 33 $\mu g \cdot kg^{-1}$ for Wilson's storm petrel, is recommended as the TRG. This guideline refers to concentration of methylmercury on a wet weight basis in the aquatic organisms (i.e., fish and other aquatic biota) that is not expected to adversely affect the wildlife that consume them. To protect all wildlife at a site, TRGs should be applied to the highest known aquatic trophic level.

For the purposes of deriving a national TRG value, the avian and mammalian RCs must be as inclusive as possible to accommodate all species and regions in Canada and are therefore based on the highest mammalian and avian FI:bw ratios known for Canadian wildlife, namely 0.24 for mink and 0.94 for Wilson's storm petrel (CCME 1998). We recognise that in areas where, for example, Wilson's storm petrel is not found, the use of the lowest RC (highest FI:BW ratio) may not be appropriate; in which case, RCs for resident wildlife may be applied. For this reason, a list of suggested RCs for a suite of mammalian and avian receptors is provided (see Table 12).

The recommended MeHg TRG of 33 $\mu g \cdot kg^{-1}$ diet, ww is lower than most MeHg concentrations that have been measured in sport fish from Canadian freshwaters (see chapter 7; Appendix B-3). Concern about human consumption advisories has led to the testing of commercially important species. Fish typically consumed by wildlife are much smaller and are from lower trophic levels, and could have proportionately less Hg than their larger commercial sport fish counterparts. As such, the reader is cautioned about making inferences regarding risks to wildlife from mercury levels in large (>35 cm) fish. The wildlife TRG, including any calculated site-specific values, must be relatively low due to the high proportion of fish in the diet of the species in question.

Table 11. TRG derivation and calculations.

STEP 1: CALCULATION OF TOLERABLE DAILY INTAKE

TDI_{AVIAN} = (LOAEL X NOAEL)$^{0.5}$ / Uncertainty factor

Where:

LOAEL = 0.48 mg·kg^{-1} food X 0.156 kg food·kg^{-1} bw

= 75 µg·kg^{-1} bw per day (Heinz 1979)

and,

NOAEL = LOAEL/5.6 (CCME 1993)

= 13 µg·kg^{-1} bw per day

Therefore,

TDI_{AVIAN} = (75 X 13)$^{0.5}$ / 1

= 31 µg·kg^{-1} bw per day.

$TDI_{MAMMALIAN}$ = (LOAEL X NOAEL)$^{0.5}$ / Uncertainty factor

Where:

LOAEL = 0.9 mg·kg^{-1} food X 0.16 kg food·kg^{-1} bw

= 144 µg·kg^{-1} bw per day (Chamberland et al. 1996)

and,

NOAEL = 0.5 mg·kg^{-1} food X 0.16 kg food·kg^{-1} bw

= 80 µg·kg^{-1} bw per day.

Therefore,

$TDI_{MAMMALIAN}$ = (144 X 80)$^{0.5}$ / 5

= 22 µg·kg^{-1} bw per day.

STEP 2: CALCULATION OF REFERENCE CONCENTRATIONS

RC_{AVIAN} = $TDI_{AVIAN} / (FI{:}BW_{MAX})$

Where:

TDI_{AVIAN} = 31 $\mu g \cdot kg^{-1}$ bw per day

and,

$FI{:}BW_{MAX}$ = 0.94 (i.e., FI:BW for Wilson's storm petrel).

Therefore,

RC_{AVIAN} = 31 / 0.94

= 33 $\mu g \cdot kg^{-1}$ diet, ww.

$RC_{MAMMALIAN}$ = $TDI_{MAMMAL} / (FI{:}BW_{MAX})$

Where:

$TDI_{MAMMALIAN}$ = 22 $\mu g \cdot kg^{-1}$ bw per day

and,

$FI{:}BW_{MAX}$ = 0.24 (i.e., FI:BW for mink).

Therefore,

$RC_{MAMMALIAN}$ = 22 / 0.24

= 92 $\mu g \cdot kg^{-1}$ diet, ww

STEP 3:

TRG = LOWEST RC = RC_{AVIAN} = 33 $\mu g \cdot kg^{-1}$ diet, ww

Table 12. Reference concentrations (RCs; µg·kg-1 diet, ww)a,b for Canadian wildlife species derived from the lowest avian (31 µg·kg-1 bw per day) or mammalian (22 µg·kg-1 bw per day) tolerable daily intakes (TDIs) for methylmercury and food intake (FI) to body weight (BW) ratiosc.

Species	FI:BW Ratio	RC
Birds		
Anseriformes		
Buffle head (*Bucephala albeola*)		
Male	0.36	86
Female	0.42	74
Common goldeneye (*Bucephala clangula*)		
Male	0.29	107
Female	0.31	100
Mallard (*Anas platyrhynchos*)	0.23	135
Oldsquaw (*Clangula hyemalis*)		
Male	0.29	107
Female	0.31	100
Wood duck (*Aix sponsa*)		
Male	0.34	91
Female	0.35	89
American wigeon (*Anas americana*)		
Male	0.32	97
Female	0.32	97
Lesser Scaup (*Aythya affinis*)		
Male	0.31	100
Female	0.32	97
Common merganser (*Mergus merganser*)		
Male	0.24	129
Female	0.27	115
Red-breasted merganser (*Mergus serrator*)		
Male	0.21	148
Falconiformes		
Bald eagle (*Haliaeetus leucocephalus*)	0.11	282
Osprey (*Pandion haliaetus*)	0.2	155
Coraciiformes		
Belted kingfisher (*Ceryle alcyon*)	0.5	62
Gaviiformes		
Common loon (*Gavia immer*)	0.18	172
Charadriiformes		
Common tern (*Sterna hirundo*)	0.61	51
Herring gull (*Larus argentatus*)		
Male	0.28	111
Female	0.29	107
Ring-billed gull (*Larus delawarensis*)		
Male	0.17	182
Black-legged kittiwake (*Rissa tridactyla*)		
Male	0.38	82
Razorbill (*Alca torda*)	0.32	97
Common murre (*Uria aalge*)		
Male	0.39	79
Female	0.3	103
Thick-billed murre (*Uria lomvia*)	0.3	103
Black guillemot (*Cepphus grylle*)	0.4	78
Atlantic puffin (*Fratercula arctica*)	0.39	79
Tufted puffin (*Fratercula cirrhata*)	0.32	97
Birds		
Ciconiiformes		
Great blue heron (*Ardea herodias*)		
Male	0.21	148
Female	0.22	141
Green-backed heron (*Butorides striatus*)	0.24	129
Procellariiformes		
Wilson's storm-petrel (*Oceanites oceanicus*)	**0.94**	**33**
Fork-tailed storm-petrel (*Oceanodroma furcata*)	0.73	42
Northern fulmar (*Fulmarus glacialis*)		
Male	0.34	91
Female	0.38	82
Mammals		
Mustelidae		
Sea otter (*Enhydra lutris*)		
Male	0.18	122
Female	0.2	110
American mink (*Mustela vison*)		
Female	0.24	92
River Otter (*Lutra canadensis*)	0.1	220
Pinnipedia		
Harbour Seal (*Phoca vitulina*)		
Male	0.16	138
Female	0.17	129
Northern fur seal (*Callorhinus ursinus*)		
Male	0.13	169
Female	0.18	122
Northern elephant seal (*Mirounga angustirostris*)		
Male	0.08	275
Female	0.1	220
Northern seal lion (*Eumetopias jubata*)		
Male	0.1	220
Female	0.12	183
Walrus (*Odobenus rosmarus*), eastern Arctic race		
Male	0.11	200
Female	0.11	200
Walrus (*Odobenus rosmarus*), Pacific Ocean race		
Male	0.1	220
Female	0.1	220
Ursidae		
Polar Bear (*Ursus maritimus*)		
Male	0.12	183

Notes:

[a] Reference Concentration refers to the concentration of methylmercury in aquatic biota that when consumed by wildlife is not expected to result in adverse effects.

[b] US EPA ranked Hg susceptibility as: kingfisher > river otter > loon = osprey = mink > bald eagle.

[c] FI: BW values from CCME (1998)

11.0 DATA GAPS

Research on methylmercury has lagged behind research on inorganic mercury because of former limitations in sample collection and analytical methodologies. As a result, a number of data gaps exist. Knowledge of specific environmental variables that influence the mercury cycle, biological accumulation, and toxicity are also contradictory. Other data gaps relate to essential toxicity and physiological studies on critical wildlife species. More information is required in the following areas:

- The influence of abundance and range of DOC types on MeHg bioavailability and accumulation by aquatic organisms;
- What levels of dietary selenium are protective, and how much of a role do internal demethylating pathways play in protecting piscivorous birds and mammals;
- Dietary absorption efficiency of MeHg from natural sources for the key piscivorous wildlife species;
- Studies that analyse for both total and MeHg so that differences between aquatic systems can be definitively linked to differences in MeHg levels. Analysing the two mercury species together will contribute to an understanding of existing data, much of which has been reported as total mercury;
- Data on MeHg levels in wildlife are needed to identify populations that are potentially at risk. Feathers and fur hold promise in this regard due to the potential for "non-invasive" determination of mercury residues;
- Whenever possible, tissue samples should be analysed for selenium. Sampling efforts with wildlife for Se and MeHg should be accompanied by analyses of likely food items;
- Chronic toxicity studies are needed for representative mammalian and avian species where a NOAEL is available in addition to a LOAEL;
- Chronic studies, if possible, should be completed with piscivorous bird species such as loons, eagles, or osprey;
- Chronic toxicity studies with river otter have never been completed;
- More specific knowledge about the diets of critical wildlife is required. Fish sampling efforts are frequently focused on species that are relevant to human consumers but that may be of little significance to wildlife. **There is an additional need to collect information for small fish (<35 cm), macroinvertebrates, amphibians and reptiles.** Seasonal and spatial effects on predation should be explored and methods developed to describe this information adequately. Additional life history data is needed to fully characterise the nature and extent of exposure to MeHg. Complicating factors must be considered, including migratory behaviours and sex-specific differences in distribution and resource allocation; and
- As the above data gaps are filled, it may be possible to extrapolate from effects of MeHg on individuals (as described in this report) to populations, communities and ecosystems.

12.0 GUIDELINE IMPLEMENTATION

For substances that have a strong potential to biomagnify through the food chain, proper implementation of the TRGs requires consideration of several factors including: 1) the identification of the wildlife species requiring protection, 2) food intake: body weight ratios and diet preferences of those wildlife species, and 3) size class and trophic level of prey (diet) items on which the species of concern feeds. Implementation of TRG for MeHg by risk managers may be particularly challenging given the toxicity of methylmercury, its widespread occurrence due to both natural and anthropogenic sources, and possible adverse effects in wild populations (Wren et al. 1986; Wobeser et al. 1976a,b; G. Mierle 1999, MOE, pers. com.). The apparent stringency of this guideline is due in part to the small size and high food intake rate of the avian species on which it is based, the Wilson's storm petrel.

To protect all wildlife species, the TRG applies broadly to the highest known aquatic trophic level (CCME 1998). Environment Canada and the CCME cautiously encourage jurisdictions to modify the guidelines into site-specific objectives where necessary. This process may include taking into account species which are protected by the national guideline but which are not present, or are not reasonably expected to be present, in the sites under consideration. Guidelines may be modified also if factors affecting toxicity or bioavailability are adequately known. For example, hardness and DOC make metals less toxic to aquatic life under some conditions and guideline modification can take this into account. More specifics on guideline modification are provided in the CCME document entitled "*Scientific and Technical Guidance on Water Quality Guideline Implementation*"(in press). Once a suitable site-specific tissue residue objective is determined, it must be applied to the appropriate dietary items (prey species), in terms of both size and trophic level.

12.1 Use of Appropriate Wildlife Species

The TRG was calculated from a tolerable daily intake (based on the toxicity of mercury to mallard ducks), and the highest food intake: body weight ratio for wildlife species, namely that for Wilson's storm petrel (see chapter 10). Wilson's storm petrel, as a marine bird, is not present in all jurisdictions and, therefore, may not be representative of resident species in freshwater ecosystems. Moreover, large birds, such as bald eagles, with small food intake: body weight ratios may tolerate a more highly contaminated diet than small birds such as terns which eat more food per body weight (i.e., large food intake: body weight ratios), assuming all other factors equal. For this reason a list of food intake: body weight ratios and corresponding reference concentration values are provided for a number of wildlife species (Table 12). Reference concentrations may themselves be modified, for example, by adjusting the food intake: body weight ratio on a site-specific basis for a given wildlife species.

12.2 Application of TRG to Appropriate Size Class of Dietary Items

Different wildlife species consume different size classes of fish species. For example, mink typically consume fish 15 to 20 cm in length while heron consume fish 20 to 25 cm in length. Generally, wildlife do not consume fish longer than 35 cm (U.S. EPA 1997c). As a result of bioaccumulation and food chain transfer, larger fish of a given species commonly contain higher methylmercury tissue concentrations than smaller fish; therefore, the TRG or site-specific objective should be applied to the size class of fish (prey) on which the wildlife species of concern feeds. Otherwise, erroneous conclusions may be drawn regarding the potential for adverse effects in wildlife consumers of aquatic biota. Notwithstanding, wildlife species that scavenge for food consume diet items of various sizes that are sometimes in excess of 35 cm.

Ultimately, reference concentrations should be compared to the concentrations in known prey and other diet items, regardless of size class or source.

12.3 Trophic Level Considerations

To develop site-specific tissue residue objectives, the trophic level at which a wildlife species feeds needs to be considered to ensure adequate protection of the resident wildlife consumers of aquatic biota. For example, a site-specific objective designed to protect wildlife that consume trophic level 2 species (e.g., common goldeneye) may not be protective of wildlife that prey on trophic level 3 species (e.g., cormorants). The following sections and Tables 10 and 11 are intended to provide general guidance necessary for the proper implementation of TRGs for bioaccumulative substances. The classification for aquatic trophic levels (Table 13; from US EPA 1995e) provides guidance on the aquatic organisms found at each trophic level, as well as the wildlife species that feed at each aquatic trophic level in freshwater environments. Feeding habits for several consumers of aquatic life are presented in Table 14 to aid guideline users in choosing an appropriate trophic level at which to apply the guideline to protect the wildlife species of concern.

12.3.1 Uncertainties with Establishing Distinct Trophic Levels

A major difficulty with the trophic levels described above is that many species feed at more than one trophic level and therefore do not fit wholly into discrete trophic levels. While species can be fairly readily identified as plants or herbivores, the carnivorous species are difficult to categorise into distinct trophic levels. For example, lake trout will consume both benthic invertebrates (trophic level 2) and small fish (trophic level 3). Mink will feed on muskrats (which feed mainly on emergent vegetation, therefore trophic level 1), shiners (trophic level 3), and some walleye (trophic level 4). In general, the closer a species is to the top of a food web, the more likely it is to feed on prey from more than one trophic level (US EPA 1995e). To overcome the uncertainty in assigning the diet of a wildlife species to a specific trophic level, the highest trophic level at which a species feeds was used for its classification.

Uncertainty also exists when attempting to estimate the food chain or web that supports a given wildlife species in a specific location. First, a food chain analysis implies a certain consistency in feeding patterns over time. The diets of animals, however, vary both with season because of changes in nutritional needs and availability of prey, and with organism age and size as larger animals can take larger prey. Thus, analysis of a particular food chain or web for an organism over a short period of time may not be indicative of the potential for bioaccumulation and biomagnification over longer time periods.

Finally, ecosystem types that would otherwise be similar (e.g., oligotrophic lakes of a certain size) may support substantially different food webs and numbers of trophic levels to the top predators depending on the history of the ecosystem, species introductions, and species loss. It has been shown that much of the large between-lake differences in contaminant levels among fish species results from differences in the length of the food chains (Rasmussen et al. 1990). This underlines the importance of understanding local food webs if the TRGs are being applied on a site-specific basis.

12.3.2 Basic Aquatic Trophic Levels

General trophic level guidance for freshwater ecosystems is provided in Table 13. Each trophic level has a general description of the species that feed within that trophic level and what their diet resembles. This table is not intended to replace best scientific judgement at a given site.

12.3.3 Prey Trophic Levels of Representative Species

Information on the feeding habits and prey trophic levels of several species in both freshwater and marine ecosystems is presented in Table 14. Again, this table is only meant for guidance and is not intended to replace best scientific judgement at a given site.

Table 13. Basic freshwater aquatic trophic levels.

Trophic Level	Organisms	Examples
1	**Primary Producers**	
	Phytoplankton	Free-floating unicellular algae.
	Periphyton	Algae attached to substrates (e.g., rocks, mud, surfaces of aquatic macrophytes).
	Emergent macrophytes	Marsh plants rooted in the littoral zone with leaves and stems exposed (e.g., cattails and bulrushes).
	Floating aquatic macrophytes	Higher plants that are free-floating and not rooted to a substrate (e.g., duckweed, pondweed, water hyacinth).
	Submerged aquatic macrophytes	Higher plants rooted to a substrate in the littoral zone that are submerged underwater (e.g., *Hydrilla*).
	Detritus	Dead and decaying plant and animal particulate matter. It may float or settle to the bottom.
2	**Herbivores and Detritivores**	
	Planktivores	Generally are zooplankton that filter phytoplankton and floating detritus and bacteria from the water column.
	Order Anostraca	Fairy shrimp.
	Conchostraca	Clam shrimp.
	Cladocera	Water fleas (e.g., *Daphnia*).
	Copepoda	Copepods.
	Diptera	Aquatic larvae of several species of flies.
	Class Bivalvia	Mussels and clams.
	Periphyton and Macrophyte Consumers	Herbivorous invertebrates that feed on the periphyton (and associated detritus) on the surfaces of plants and other aquatic substrates, or that feed on macroalgae and higher plants.
	Order Amphipoda	Scuds, side-swimmers, or freshwater shrimp.
	Decapoda	Crayfish.
	Coleoptera	Haliplidae (crawling water beetles).
	Diptera	Aquatic larvae of flies and midges.
	Lepidoptera	Caterpillars of butterflies and moths
	Gastropoda	Snails.
	Class Amphibia	Tadpoles, larval newts.
	Bony fishes	Several species including lake herring, shad, and carp.
	Detritivores	Scavenge food from substrates or through filter-feeding.
	Order Copepoda	Copepods.
	Ostracoda	Seed shrimp.
	Amphipoda	Pontoporeia, scuds, side-swimmers, or freshwater shrimp.
	Diptera	Aquatic larvae of midges and several species of flies.
	Decapoda	Grass shrimp.
3	**Small Carnivores**	
	Invertebrate Carnivores	
	Order Caldocera	*Leptodora* spp., *Polyphemus* spp.
	Mysidae	*Mysis* spp.
	Coleoptera	Dytiscidae (predatory diving beetles).
	Hemiptera	Aquatic bugs such as water bugs, water boatmen, and water striders.
	Odonata	Dragonfly and damselfly nymphs and adults.
	Ephemeroptera	Nymphs of some mayflies.

Trophic Level	Organisms	Examples
	Invertebrate Omnivores	Many groups of invertebrates include species that feed on both plant and animal matter or that display a wide variety of dietary habits among species. One of the more ubiquitous of these groups are the Trichopterans (or caddis flies). This group includes many omnivorous species, but some species are exclusively grazers, scrapers, suspension feeders, filter feeders, or carnivores.
	Small Vertebrate Carnivores	Species that feed on zooplankton and benthic invertebrates.
	Fish species	Forage fish species including shiners (up to 5 to 6 cm), mudminnows, darters, killifish, sticklebacks, longnose dace, mummichog, suckers, alewife, smelt, sculpin, chub, crappie, bullheads, sunfish, white perch, yellow perch (until age 4), carp.
4	**Large Carnivores**	
	Predacious fish species	The larger carnivores, or predators, can feed on the smaller carnivores, but also tend to feed on the larger herbivorous species. In freshwater aquatic ecosystems, the larger carnivores are exclusively fish. Examples of key species include trout (particularly greater than 30 cm), salmon, walleye, northern squawfish (> 30 cm), burbot (> 50 cm), redfin and chain pickerel, coho salmon, Arctic char (> 20 cm), channel catfish (> 45 cm), gar, and pike.

Source: US EPA 1995e

Table 14. Feeding habits and prey trophic levels of representative freshwater amphibian, reptilian, avian, and mammalian species.

Species	Prey Trophic Level	Feeding Habits
Amphibians		
Frogs	2	Capture insects that may have an aquatic or terrestrial food chain base.
Salamanders	2	Adults and nymphs are carnivorous.
Mudpuppies	2	Feed on worms, crayfish, insects, and small fish.
Reptiles		
Water snakes	3	Feed primarily on small fish and frogs.
Common snapping turtle	3	Feed primarily on aquatic invertebrates, small fish, and amphibians.
Eastern painted turtle	2	Feed on insects, snails, and bits of lily pad.
Birds		
Herring gull	4	Herring gulls frequent a wide variety of coastal areas including freshwater and marine. They are highly opportunistic feeders and feed on a wide variety of foods depending on availability including fish, squid, crustaceans, molluscs, worms, insects, small mammals and birds, duck and gull eggs and chicks, and garbage. Freshwater fish usually considered trophic level 3 (up to 23 cm in length, i.e., small freshwater drum, alewife, smelt) comprise a large proportion of herring gull diets in most populations, although some trophic level 4 fish are also taken. About 75% of their diet is fish.
Bald eagle	4	Bald eagles are generally found in coastal areas, lakes, and rivers. They will eat dead or dying fish over most of their range, but also catch live fish swimming near the surface and often eat mammals and birds. In general, bald eagles can be described as opportunistic feeders, eating whatever food source is most plentiful and easy to capture. Bald eagles can capture and carry relatively large prey (i.e., > 60 cm in length) such as chain pickerel, burbot, and lake trout.
Belted kingfisher	3	Belted kingfishers are typically found along rivers and streams and along lake and pond edges. They are also common on sea coasts and estuaries. Kingfishers feed predominantly on fish, although they sometimes consume large numbers of crayfish. The largest fish prey taken are generally less than 18 cm, the average length being closer to 5 to 8 cm. Freshwater species known to be captured by kingfishers include brook trout, sculpins, blacknose dace, creek chub, common shiner, darters, brook stickleback, redbelly dace, fathead minnow, and suckers.
Osprey	3	Osprey are found near fresh or salt water and are almost completely piscivorous. Although, they have been observed on occasion to take other prey including birds, frogs, and crustaceans. Freshwater fish prey include gizzard shad, yellow perch, and salmonids. Most of the fish captured by osprey are between 10 and 35 cm in length.
Ring-billed gull	3	Although smaller in body size, ring-billed gulls inhabit areas similar to the herring gull. They are generalised and opportunistic foragers, consuming large quantities of fish as well as significant quantities of terrestrial, aquatic, and aerial invertebrates. A study in the Great Lakes found ring-billed gulls to consume mainly smelt, alewives, and sticklebacks. They also consumed earthworms, ephemeroptera (mayflies), homoptera (cicadas), coleoptera (beetles), and diptera (chironomid midges).
Black-crowned night heron	3	Black-crowned night herons nest in a wide variety of freshwater, brackish, and saltwater habitats. They are considered opportunistic general predators, feeding primarily on fish, amphibians, and insects (beetles, flies, and dragonfly nymphs) in freshwater habitats, and on molluscs, spiders, small mammals (e.g., voles), and birds and eggs. Fish species up to 17 cm in length have been taken and include

Species	Prey Trophic Level	Feeding Habits
		whiting, herring, carp, pickerel, suckers, horn-pouts, black bass, perch, gizzard shad, alewife, and eels.
Common tern	3	Common terns usually breed on island or coastal beach habitat. They forage in large flocks over schools of small fish, with fish comprising over 90% of their diet. They feed secondarily on crustaceans and insects. In freshwater, common terns have been found to prey on alewife, smelt, bluntnose minnow, common shiners, emerald shiners, and trout-perch.
Forster's tern	3	Forster's tern breeds in freshwater and saltwater marshes and on marshy borders of ponds and lakes. Little information exists on the feeding preferences of the Forster□s tern, but it is known that it feeds on insects as it flies over marshes and also feeds on fish. Chicks are fed minnows almost exclusively. They also eat frogs, scavenge dead fish, and occasionally take the eggs of the American coot.
Caspian tern	3	Caspian terns breed on flat sand or gravel beaches, shell banks, and occasionally marshes in both marine and freshwater. They dive primarily for fish and occasionally take crustaceans. Fish species eaten include alewife, smelt, yellow perch, pumpkinseed, and rock bass. Fish taken are usually 10-12 cm in length.
Black tern	3	Black terns breed in shallow freshwater marshes. During the breeding season, they eat mainly insects, including dragonflies, moths, grasshoppers and crickets, beetles, spiders, water scorpions, mayflies, and caddisflies, and smaller amounts of grubs, larvae, small fish, molluscs, and crayfish.
Double-crested cormorant	3	Double-crested cormorants frequent coasts, bays, estuaries, marine islands, freshwater lakes and their islands, ponds, rivers, sloughs, and swamps. Their diet consists primarily of schooling fish, but may include some small invertebrates. Double-crested cormorants often take small forage fishes such as sticklebacks, sculpins, and burbot, and also take yellow perch, white sucker, and tulibee. Most of the fish taken range from 12-15 cm in length.
Common and American mergansers	3	The American merganser is recognised by some as a subspecies of the common merganser. Common mergansers generally occur in a variety of open freshwater habitats including cold and warm water rivers, ponds, lakes, and inland bays. In riverine habitats, they have been observed to prefer trout and young salmon, and in larger bodies of water will utilise forage fish in large schools. In lakes and rivers, the mergansers were capturing small salmonids and small to medium sculpins (3 to 12 cm in length).
Red-breasted merganser	3	The habit of the red-breasted merganser is much more marine than that of the common merganser. In the breeding season, it inhabits inland waters as well as coasts and marine islands. In other seasons it inhabits tidewater and inshore marine areas. In freshwater areas, red-breasted mergansers ate salmon eggs, salmonids, sticklebacks, sculpins, schizopods (crusatcean), and caddis larvae.
Great blue heron	3	Great blue herons are found in a variety of freshwater and marine habitats, including freshwater lakes and rivers, brackish marshes, lagoons, mangroves, and coastal wetlands. They are often seen on tidal flats and sandbars, and occasionally forage in wet meadows, pastures, and other terrestrial habitats. Fish are the preferred prey, but they also eat amphibians, reptiles, crustaceans, insects, birds, and mammals. Most of the fish captured represent trophic level 3 (including horn-pouts, shiners, perch, suckers, black bass, herrings, small pickerel) and are less than 25 cm in length.
Loons	3	Loons feed primarily on small fish at trophic level 3.
Western grebe	3	The western grebe diet includes trophic level 2 and 3 aquatic invertebrates and small fish between 3 and 20 cm in length.
Lesser scaup	2	Lesser scaup are found on large lakes and bays during the fall and winter, and are common on smaller bodies of water during the spring.

Species	**Prey Trophic Level**	**Feeding Habits**
		Most populations consume primarily aquatic invertebrates year round. Common prey include snails, clams, amphipods, midges, chironomids, and leeches. In spring and summer, egg-laying females and ducklings feed at about trophic level 2.
Common goldeneye	2	During the summer on inland lakes, common goldeneyes tend to consume aquatic invertebrates (75%) and plant material (25%).
Mallard duck	2	Mallards prefer natural bottomland wetlands and rivers to reservoirs and farm ponds. They feed primarily on seeds of aquatic plants and cultivated grains, although they also consume aquatic invertebrates, particularly during the breeding season. During the summer, 80-90% of their diet is made up of aquatic invertebrates.
Snow goose	1	Snow geese are almost entirely herbivorous year-round.
Canada goose	1	Canada geese are almost exclusively vegetarian year-round. They graze on terrestrial and wetland plants.
American black duck	1	The spring and summer diet in freshwater habitat is 80% aquatic plants.
Mammals		
River otter	4	River otters are almost exclusively aquatic and are found in food rich coastal areas, the lower portions of streams and rivers, estuaries, nonpolluted waterways, the lakes and tributaries that feed rivers, and areas showing little human impact. They primarily consume fish, but may also consume crustaceans, aquatic insects (e.g., stonefly nymphs, aquatic beetles), amphibians, insects, birds (e.g., ducks), mammals, and turtles. Otters feed primarily on trophic level 3 fish, including suckers, mudminnows, shiners, darters, and carp. They also capture some perch and level 4 walleye, but very few trout and burbot. The average length of fish captured is about 13 cm.
Harbour seal	4	Some freshwater harbour seals are found in Canada. They were found to be depleting older lake trout populations in Québec, although all of the fish were less than 55 cm in length.
Mink	3	Mink are found associated with aquatic habitats of all kinds, including water ways such as rivers, streams, lakes, and ditches, as well as swamps, marshes, and backwater areas. Mink are generalist and opportunistic feeders, taking whatever prey is locally abundant. The diet of mink consists primarily of prey linked to aquatic ecosystems, including crayfish, frogs, fish, muskrat, and waterfowl. Terrestrial prey include shrews, mice, and voles. Fish species taken include trout, sculpins, blacknose dace, creek chub, suckers, darters, and redbelly dace measuring less than 18 cm in length.
Raccoon	2	Raccoons are found near virtually every aquatic habitat, particularly in hardwood swamps, mangroves, floodplain forests, and fresh and salt water marshes. Raccoons consume less fish then do river otters or mink. They consume a high amount of fruit and nuts., as well as aquatic invertebrates. Their diet rarely contains trophic level 3 fish.

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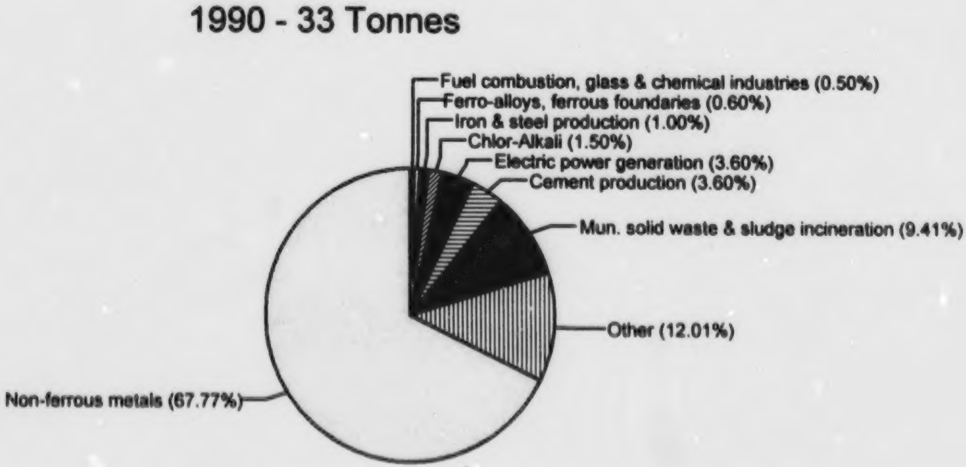


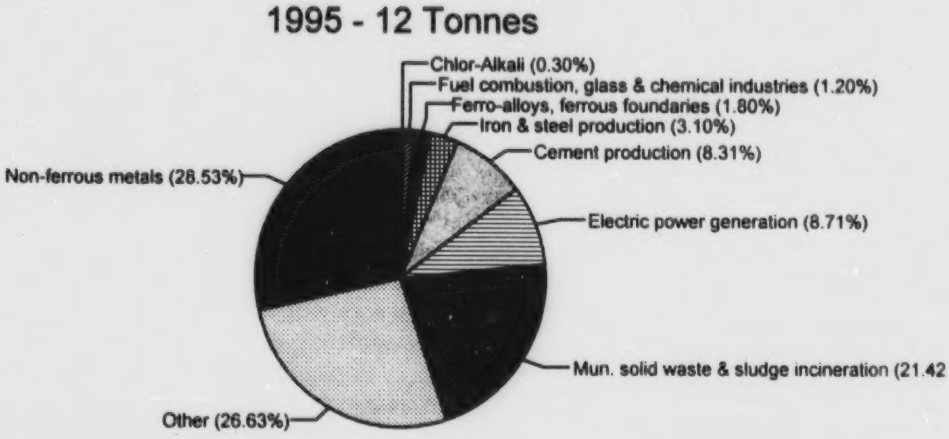
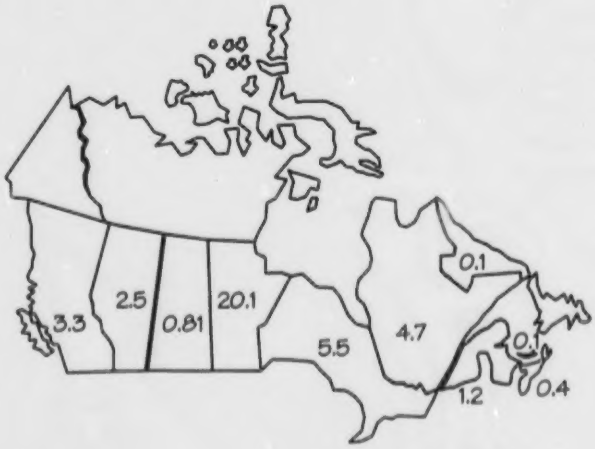


Figure 1. **Mercury emissions in Canada by sector for 1990 and 1995 as estimated by the Transboundary Air Issues Branch of Environment Canada.**

Source: Environment Canada 1997; L. Trip, unpubl. data



Note that Manitoba's emissions were later reduced by 20 T.

Figure 2. Tonnes of mercury emitted by each Canadian province in 1990 as estimated by the Transboundary Air Issues Branch of Environment Canada.

Source: Pilgrim and EMAN 1998

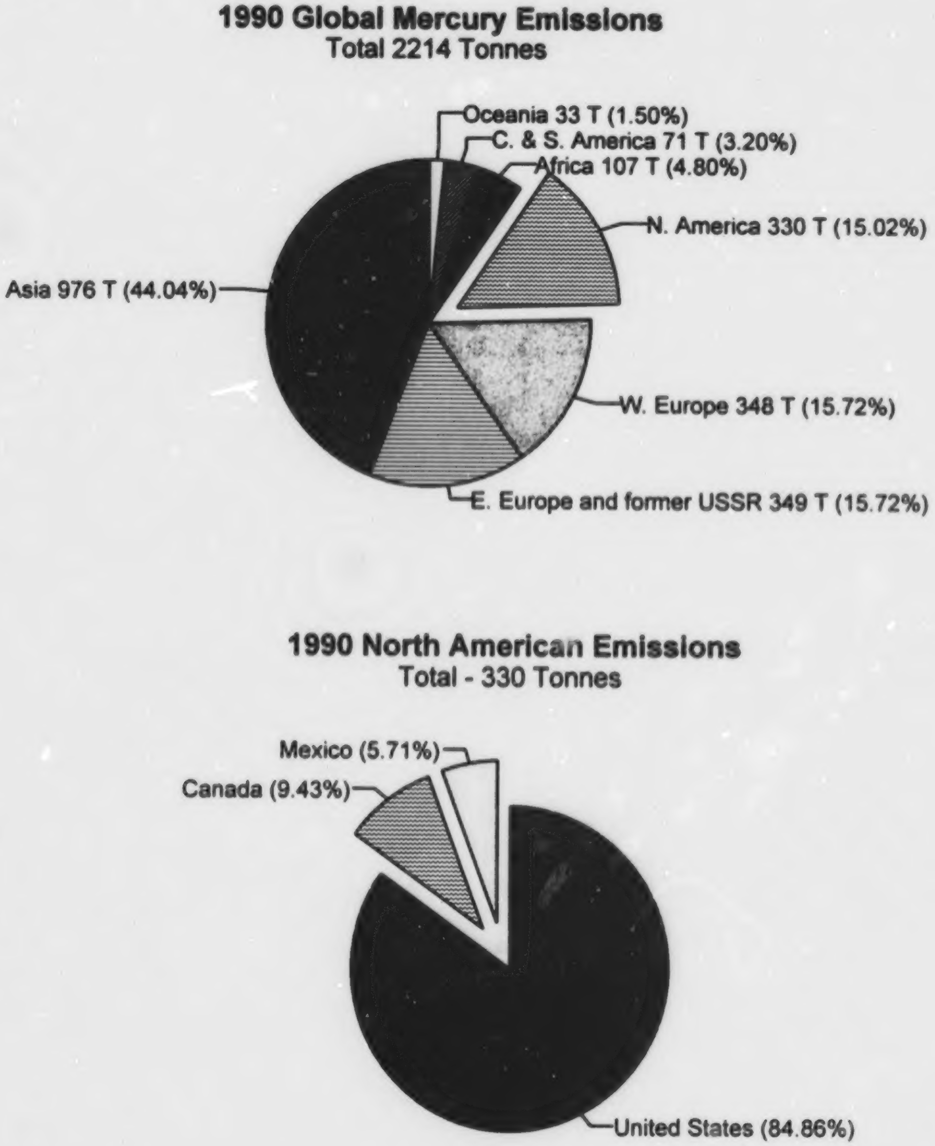


Figure 3. **Global and North American total mercury emissions for 1990 as reported by the Transboundary Air Issues Branch of Environment Canada.**

Source: Environment Canada 1997; L. Trip 1999, Environment Canada, pers. com.

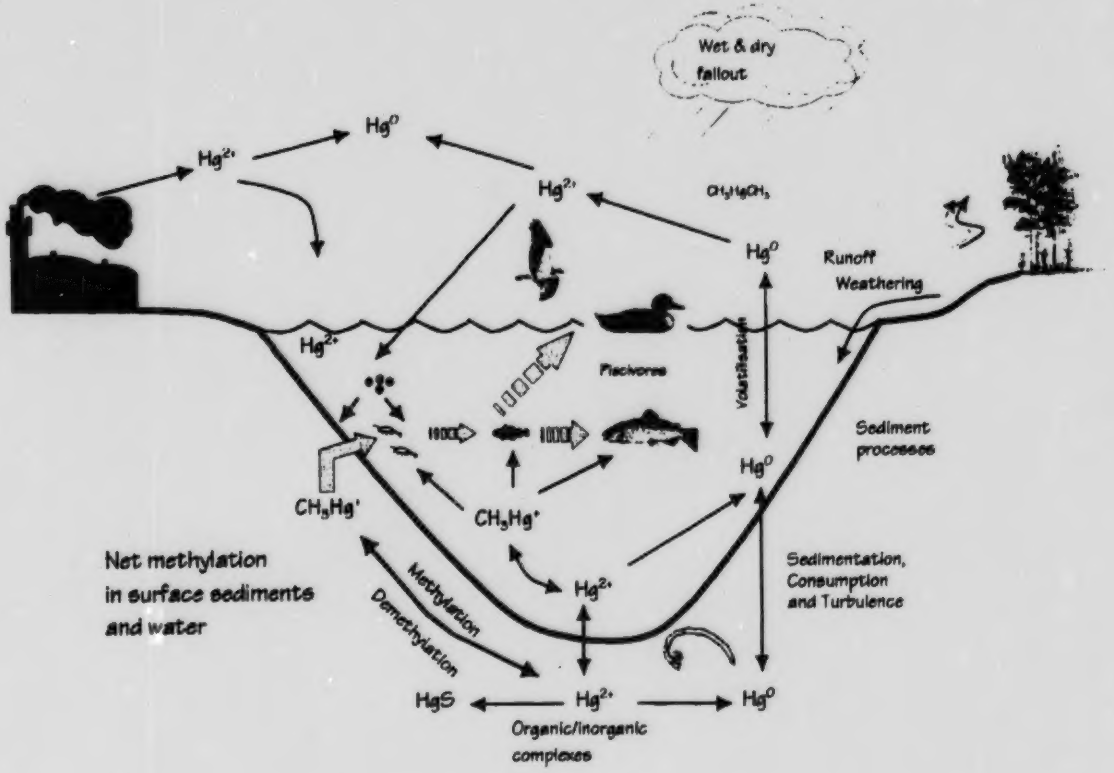

Wet & dry fallout
Hg2+
Hg0
Hg2+
CH3HgCH3
Hg0
Runoff
Weathering
Hg2+
Piscivores
Volatilisation
Sediment processes
Hg0
CH3Hg+
CH3Hg+
Net methylation in surface sediments and water
Methylation
Demethylation
Hg2+
Sedimentation, Consumption and Turbulence
HgS
Hg2+
Hg0
Organic/inorganic complexes

Figure 4. **The Mercury Cycle**

APPENDIX A: CANADIAN STRATEGIES UNDER A BILATERIAL STRATEGY TO EVENTUALLY ELIMINATE ANTHROPOGENIC TOXIC SUBSTANCES IN THE GREAT LAKES BASIN.

In Canada:

It has been estimated that between 2 700 and 3 450 kg of mercury are released to the atmosphere in Ontario annually from anthropogenic sources, while up to 2 500 kg are released to the waters of the Great Lakes Basin annually. Through an analysis of mercury uses and sources, significant sources of mercury have been identified and prioritised. These sources will be encouraged to develop strategies to reduce their releases by 90 percent from a baseline year of 1988 through adoption of pollution prevention measures.

In partnership with Pollution Probe, Canada and Ontario have identified potential industrial partners to participate in a unique three-way initiative to reduce or eliminate mercury in industrial or commercial applications. Co-ordination of this effort with U.S. partners is being considered, and the findings and approaches are being shared with the U.S. Virtual Elimination Pilot Project.

Activities by companies to date have resulted in significant reductions in mercury content in batteries (60 - 90 percent), fluorescent lamps (44 percent) and switches, while further reductions are planned, such as 70 percent by fluorescent lamp manufacturers by 2000. One impact of past mercury usage is that landfill emissions may be a source of mercury releases in the Great Lakes Basin, but the quantities released and possible control mechanisms need further consideration.

In applying the analytical framework in addressing mercury, relevant information from research projects undertaken by Environment Canada, Natural Resources Canada, and other agencies will be considered.

Canada will work with the U.S. and Mexico in implementing the North American Regional Action Plan for Mercury and will incorporate mercury reduction targets in its partnerships with commercial and industrial sectors in Ontario (Environment Canada and US EPA 1997).

Source: Environment Canada and US EPA 1997

APPENDIX B-1: LEVELS OF TOTAL MERCURY AND METHYLMERCURY IN FRESHWATER AQUATIC PLANTS IN THE CANADIAN ENVIRONMENT.

Location	Year	Species	Tissue	N	Total Hg μg·kg^{-1} dw	MeHg μg·kg^{-1} dw	% MeHg	Reference
BC: Nahounli L.	1982	Macrophyte *Myriophyllum spicatum*	Shoot	8	38 ± 6	NR	NR	Siegel et al. 1985
BC: Nahounli L.	1982	Macrophyte *Potamogeton* spp.	Shoot	8	61 ± 11	NR	NR	Siegel et al. 1985
BC: Pinchi L.	1982	Macrophyte *Myriophyllum spicatum*	Shoot	8	37 to 176	NR	NR	Siegel et al. 1985
BC: Pinchi L.	1982	Macrophyte *Potamogeton* spp.	Shoot	8	15 to 132	NR	NR	Siegel et al. 1985
ON: Bentshoe L. Muskoka-Haliburton	1988	Macrophyte *Eriocaulon septangulare*	Root	13	90 to 130	NR	NR	Coquery and Welbourn 1995
ON: Bentshoe L. Muskoka-Haliburton	1988	Macrophyte *Eriocaulon septangulare*	Leaf	13	40 to 110	NR	NR	Coquery and Welbourn 1995
ON: Chub L. South-central Ontario	1981	Filamentous Algae *Mougeotia* spp. And *Spirogyra* spp.	Whole	3	90 to 330	NR	NR	Stokes et al. 1983
ON: Crosson L. South-central Ontario	1981	Filamentous Algae *Mougeotia* spp. And *Spirogyra* spp.	Whole	3	40 to 180	NR	NR	Stokes et al. 1983
ON: Dickie L. South-central Ontario	1981	Filamentous Algae *Mougeotia* spp. and *Spirogyra* spp.	Whole	2	120; 120	< 50	NR	Stokes et al. 1983
ON: Experimental Lakes Area	1992	Macrophyte/sedge *Carex aquatilis, C. rostrata,* and *Sparganium spp.*	Whole	4	6 to 18.8	1.3 to 6.4	37	Moore et al. 1995

Location	Year	Species	Tissue	N	Total Hg µg·kg^{-1} dw	MeHg µg·kg^{-1} dw	% MeHg	Reference
ON: Experimental Lakes Area Hummocks	1992	Moss/bryophyte *Sphagnum* spp.	Whole	2	36.3 ± 10.7	0.45 ± 0.35	1.4	Moore et al. 1995
ON: Experimental Lakes Area Hollow/lawn	1992	Moss/bryophyte *Sphagnum* spp.	Whole	5	46.7 ± 18.8	1.02 ± 0.63	2.5	Moore et al. 1995
ON: Experimental Lakes Area Pool/stream	1992	Moss/bryophyte *Sphagnum* spp.	Whole	2	92.5 ± 38.1	20.4 ± 22.1	19	Moore et al. 1995
ON: Experimental Lakes Area	1992	Moss/bryophyte *Sphagnum augustifolium*	Capitulum	NR	91.9	2.5	2.7	Moore et al. 1995
ON: Experimental Lakes Area	1992	Moss/bryophyte *Sphagnum augustifolium*	Upper Stem	NR	116.8	1.6	1.4	Moore et al. 1995
ON: Experimental Lakes Area	1992	Moss/bryophyte Sphagnum augustifolium	Lower Stem	NR	125.3	0.3	0.3	Moore et al. 1995
ON: Fawn L. South-central Ontario	1981	Filamentous Algae Mougeotia spp. and Spirogyra spp.	Whole	2	87; 96	NR	NR	Stokes et al. 1983
ON: Harp L. South-central Ontario	1981	Filamentous Algae *Mougeotia* spp. and *Spirogyra* spp.	Whole	2	160; 220	NR	NR	Stokes et al. 1983
ON: Heney L. South-central Ontario	1981	Filamentous Algae *Mougeotia* spp. and *Spirogyra* spp.	Whole	3	80 to 93	50	55	Stokes et al. 1983
ON: Ottawa R., near Ottawa	1975	Macrophyte *Elodea canadensis*	Shoot	2	65 to 225	NR	30	Mortimer 1985
ON: Ottawa R., near Ottawa	1975	Macrophyte *Elodea canadensis*	Root	2	159 to 260	NR	NR	Mortimer 1985
ON: Ottawa R., near Ottawa	1976	Macrophyte *Elodea canadensis*	Shoot	6	NR	NR	24	Mortimer 1985

Location	Year	Species	Tissue	N	Total Hg µg·kg^{-1} dw	MeHg µg·kg^{-1} dw	% MeHg	Reference
ON: Ottawa R., near Ottawa	1976	Macrophyte *Elodea canadensis*	Root	6	NR	NR	11	Mortimer 1985
ON: Ottawa R., near Ottawa	1975	Macrophyte *Phalaris arundinacea*	Shoot	5	79 to 254	NR	NR	Mortimer 1985
ON: Ottawa R., near Ottawa	1975	Macrophyte *Phalaris arundinacea*	Root	4	211 to 448	NR	NR	Mortimer 1985
ON: Ottawa R., near Ottawa	1975	Macrophyte *Sagittaria latifolia*	Shoot	4	54 to 94	NR	39	Mortimer 1985
ON: Ottawa R., near Ottawa	1975	Macrophyte *Sagittaria latifolia*	Root	4	111 to 207	NR	9	Mortimer 1985
ON: Ottawa R., near Ottawa	1976	Macrophyte *Sagittaria latifolia*	Shoot	9	NR	NR	27	Mortimer 1985
ON: Ottawa R., near Ottawa	1976	Macrophyte *Sagittaria latifolia*	Root	9	NR	NR	12	Mortimer 1985
ON: Ottawa R., near Ottawa	1975	Macrophyte *Sparganium augustifolium*	Shoot	3	71 to 215	NR	30	Mortimer 1985
ON: Ottawa R., near Ottawa	1975	Macrophyte *Sparganium augustifolium*	Root	3	97 to 252	NR	14	Mortimer 1985
ON: Ottawa R., near Ottawa	1976	Macrophyte *Sparganium augustifolium*	Shoot	5	NR	NR	33	Mortimer 1985
ON: Ottawa R., near Ottawa	1976	Macrophyte *Sparganium augustifolium*	Root	8	NR	NR	8	Mortimer 1985
ON: Ottawa R., near Ottawa	1975	Macrophyte *Sparganium eurycarpum*	Shoot	3	50 to 164	NR	48	Mortimer 1985
ON: Ottawa R., near Ottawa	1975	Macrophyte *Sparganium eurycarpum*	Root	3	48 to 550	NR	11	Mortimer 1985

Location	Year	Species	Tissue	N	Total Hg µg·kg^{-1} dw	MeHg µg·kg^{-1} dw	% MeHg	Reference
ON: Ottawa R., near Ottawa	1976	Macrophyte *Sparganium eurycarpum*	Shoot	4	NR	NR	23	Mortimer 1985
ON: Ottawa R., near Ottawa	1976	Macrophyte *Sparganium eurycarpum*	Root	4	NR	NR	8	Mortimer 1985
ON: Plastic L. South-central Ontario	1981	Filamentous Algae *Mougeotia* spp. and *Spirogyra* spp.	Whole	2	160; 180	NR	NR	Stokes et al. 1983
ON: Red Chalk L. South-central Ontario	1981	Filamentous Algae *Mougeotia* spp. and *Spirogyra* spp.	Whole	1	270	NR	NR	Stokes et al. 1983
ON: St. Lawrence R., near Cornwall	1994	Macrophyte *Elodea canadensis*	Shoot	40	58 to 225	NR	NR	Thompson-Roberts et al. 1999
ON: St. Lawrence R., near Cornwall	1994	Macrophyte *Myriophyllum spicatum*	Shoot	55	63 to 240	NR	NR	Thompson-Roberts et al. 1999
ON: St. Lawrence R., near Cornwall	1976	Macrophyte *Myriophyllum spicatum*	Shoot	NR	68 to 247	NR	0 to 30 %	Mortimer 1985
ON: St. Lawrence R., near Cornwall	1978	Macrophyte *Myriophyllum spicatum*	Shoot	NR	17 to 1600	NR	0 to 30 %	Mortimer 1985
ON: St. Lawrence R., near Cornwall	1994	Macrophyte *Nuphar variegatum*	Leaf Petiole	11 5	8 to 21 6 to 37	NR	NR	Thompson-Roberts et al. 1999
ON: St. Lawrence R., Near Cornwall	1994	Macrophyte *Potamogeton crispus*	Shoot	15	80 to 85	NR	NR	Thompson-Roberts et al. 1999
ON: St. Lawrence R., Near Cornwall	1975	Macrophyte *Sagittaria latifolia*	Shoot Root	NR	NR	NR	38.6 8.7	Mortimer 1985

Location	Year	Species	Tissue	N	Total Hg µg·kg^{-1} dw	MeHg µg·kg^{-1} dw	% MeHg	Reference
ON: Swan L. South-central Ontario	1981	Filamentous Algae *Mougeotia* spp. and *Spirogyra* spp.	Whole	2	120; 130	< 50	NR	Stokes et al. 1983
ON: Wetlands (22) South-central Ontario	1993	Macrophyte *Nuphar variegatum*	Leaf Petiole	11 0	13 to 29 14 to 31	NR	NR	Thompson 1996

NR = Not reported

APPENDIX B-2A: LEVELS OF TOTAL MERCURY AND METHYLMERCURY IN FRESHWATER INVERTEBRATES IN THE CANADIAN ENVIRONMENT.

Location	Year	Species	Life Stage or Size	Tissue	N	Total Hg $\mu g \cdot g^{-1}$ ww^a	MeHg $\mu g \cdot g^{-1}$ ww^a	% MeHg	Reference
MB: Mynarksi L. Notigi Reservoir	1981	Chironomids	Larvae	Whole	3	0.014 - 0.025	NR	NR	Jackson 1988b
MB: Mynarksi L. Notigi Reservoir	1981	Clams *Pelecypoda*	Adult	Whole	1	0.015	NR	NR	Jackson 1988b
MB: Mynarksi L. Notigi Reservoir	1981	Earthworms *Oligochaeta*	Adult	Whole	1	0.337	NR	NR	Jackson 1988b
MB: Mynarksi L. Notigi Reservoir	1981	Roundworms *Nematoda*	Adult	Whole	1	0.164	NR	NR	Jackson 1988b
MB: Notigi L. Notigi Reservoir	1981	Chironomids	Larvae Pupae	Whole	1	0.024 0.026	NR	NR	Jackson 1988b
MB: Notigi L. Notigi Reservoir	1981	Earthworms *Oligochaeta*	Adult	Whole	1	0.051	NR	NR	Jackson 1988b
MB: Notigi L. Notigi Reservoir	1981	Roundworms *Nematoda*	Adult	Whole	1	0.032	NR	NR	Jackson 1988b
MB: Rat L. Notigi Reservoir	1981	Chironomids	Larvae	Whole	4	0.017 - 0.028	NR	NR	Jackson 1988b
MB: Rat L. Notigi Reservoir	1981	Clams *Pelecypoda*	Adult	Whole	1	0.078	NR	NR	Jackson 1988b
MB: Rat L. Notigi Reservoir	1981	Earthworms *Oligochaeta*	Adult	Whole	1	0.043	NR	NR	Jackson 1988b
MB: Rat L. Notigi Reservoir	1981	Roundworms *Nematoda*	Adult	Whole	1	0.097	NR	NR	Jackson 1988b
MB: Southern Indian L. Long Bay – shallow	1981	Chironomids	Larvae	Whole	1	0.208	NR	NR	Jackson 1988b
MB: Southern Indian L. Long Bay – shallow	1981	Clams *Pelecypoda*	Adult	Whole	1	0.307	NR	NR	Jackson 1988b

Location	Year	Species	Life Stage or Size	Tissue	N	Total Hg $\mu g \cdot g^{-1}$ ww^a	MeHg $\mu g \cdot g^{-1}$ ww^a	% MeHg	Reference
MB: Southern Indian L. Long Bay – shallow	1981	Earthworms *Oligochaeta*	Adult	Whole	1	0.698	NR	NR	Jackson 1988b
MB: Southern Indian L. Long Bay – shallow	1981	Roundworms *Nematoda*	Adult	Whole	1	0.459	NR	NR	Jackson 1988b
MB: Southern Indian L. South Bay	1981	Chironomids	Larvae	Whole	1	0.115	NR	NR	Jackson 1988b
MB: Southern Indian L. South Bay	1981	Clams *Pelecypoda*	Adult	Whole	1	0.023	NR	NR	Jackson 1988b
ON & QC: Lakes from 44°12N – 48°07N and 70°52W - 80°17W	1990 - 1991	Zooplankton^b - unsorted	>225 µm	Whole	24	0.108 ± 0.076 dw	NR	NR	Tremblay et al. 1995
ON: Bear L.	1986	Crayfish *Cambarus bartoni*	7.1 ± 2.2 g	Muscle	39	0.124 ± 0.044	NR	NR	Allard and Stokes 1989
ON: Blue Chalk L.	1991	Crayfish *Orconectes virilis*	2.1-3.5 cm	Muscle	11	0.022 to 0.055	NR	NR	Headon et al. 1996
ON: Clay L. East basin	1979	Crayfish *Orconectes virilis*	Adult	Muscle	20	2.2 ± 0.66	NR	NR	Parks et al. 1991
ON: Clay L. East basin	1981	Crayfish *Orconectes virilis*	Adult	Muscle	19	0.96 ± 0.34	NR	NR	Parks et al. 1991

Location	Year	Species	Life Stage or Size	Tissue	N	Total Hg $\mu g \cdot g^{-1}$ ww[a]	MeHg $\mu g \cdot g^{-1}$ ww[a]	% MeHg	Reference
ON: Cygnet L.	1980	Crayfish *Orconectes virilis*	Adult	Muscle	18	0.06 ± 0.02	NR	NR	Parks et al. 1991
ON: Dickie L.	1986	Crayfish *Cambarus robustus*	4.9 ± 5.5 g	Muscle	19	0.087 ± 0.033	NR	NR	Allard and Stokes 1989
ON: Eagle L./ Eagle R.	1980	Crayfish *Orconectes virilis*	Adult	muscle	31	0.07 ± 0.03	NR	NR	Parks et al. 1991
ON: Eagle L./ Eagle R.	1981	Crayfish *Orconectes virilis*	Adult	Muscle	6	0.06 ± 0.05	NR	NR	Parks et al. 1991
ON: Eaglenest L.	1979	Crayfish *Orconectes virilis*	Adult	Muscle	18	0.43 ± 0.12	NR	NR	Parks et al. 1991
ON: Eaglenest L.	1980	Crayfish *Orconectes virilis*	Adult	Muscle	26	0.21 ± 0.10	NR	NR	Parks et al. 1991
ON: ELARP Reservoir (L. 104)	1992	Freshwater mussel *Pyganodon grandis*	Adult	Foot	12	0.422 ± 0.033 dw	0.250 ± 0.018 dw	62%	Malley et al. 1996
ON: ELARP Reservoir (L. 104)	1992	Freshwater mussel *Pyganodon grandis*[c]	Adult	Gill	11	0.315 ± 0.044 dw	0.171 ± 0.019 dw	58%	Malley et al. 1996

Location	Year	Species	Life Stage or Size	Tissue	N	Total Hg µg·g^{-1} ww^{a}	MeHg µg·g^{-1} ww^{a}	% MeHg	Reference
ON: ELARP Reservoir (L. 104)	1992	Freshwater mussel *Pyganodon grandis*	Adult	Kidney	6	1.992 ± 0.250 dw	0.288 ± 0.023 dw	15%	Malley et al. 1996
ON: ELARP Reservoir (L. 104)	1992	Freshwater mussel *Pyganodon grandis*^{c}	Adult	Mantle	12	0.245 ± 0.014 dw	0.108 ± 0.009 dw	45%	Malley et al. 1996
ON: ELARP Reservoir (L. 104)	1992	Freshwater mussel *Pyganodon grandis*	Adult	Viscera	12	0.322 ± 0.014 dw	0.151 ± 0.009 dw	47%	Malley et al. 1996
ON: ELARP Reservoir (L. 104)	1992	Freshwater mussel *Pyganodon grandis*	Adult	Whole	12	0.368 ± 0.018 dw	0.161 ± 0.009 dw	44%	Malley et al. 1996
ON: English R., inflow to Ball L.	1979	Crayfish *Orconectes virilis*	Adult	Muscle	30	0.25	NR	NR	Parks et al. 1991
ON: English R., inflow to Ball L.	1980	Crayfish *Orconectes virilis*	Adult	Muscle	17	0.15 ± 0.06	NR	NR	Parks et al. 1991
ON: English R., inflow to Ball L.	1981	Crayfish *Orconectes virilis*	Adult	Muscle	42	0.12 ± 0.06	NR	NR	Parks et al. 1991
ON: English R., outflow of Ball L.	1979	Crayfish *Orconectes virilis*	Adult	Muscle	30	0.68 ± 0.36	NR	NR	Parks et al. 1991
ON: English R., outflow of Ball L.	1980	Crayfish *Orconectes virilis*	Adult	Muscle	18	0.42 ± 0.20	NR	NR	Parks et al. 1991

Location	Year	Species	Life Stage or Size	Tissue	N	Total Hg $\mu g \cdot g^{-1}$ ww[a]	MeHg $\mu g \cdot g^{-1}$ ww[a]	% MeHg	Reference
ON: English R., outflow of Ball L.	1981	Crayfish *Orconectes virilis*	Adult	Muscle	40	0.48 ± 0.23	NR	NR	Parks et al. 1991
ON: Four Mile L.	1986	Crayfish *Orconectes propinquus*	2.8 ± 1.1 g	Muscle	27	0.042 ± 0.018	NR	NR	Allard and Stokes 1989
ON: Four Mile L.	1986	Crayfish *Orconectes virilis*	4.9 ± 2.3 g	Muscle	14	0.095 ± 0.025	NR	NR	Allard and Stokes 1989
ON: Galeairy L.	1986	Crayfish *Orconectes obscurus*	4.4 ± 2.3 g	Muscle	31	0.073 ± 0.029	NR	NR	Allard and Stokes 1989
ON: Gull L.	1986	Crayfish *Cambarus robustus*	16.8 ± 9.7 g	Muscle	22	0.062 ± 0.036	NR	NR	Allard and Stokes 1989
ON: Kimball L.	1986	Crayfish *Cambarus bartoni*	7.3 ± 3.5 g	Muscle	43	0.089 ± 0.057	NR	NR	Allard and Stokes 1989
ON: L. Vernon	1986	Crayfish *Cambarus bartoni*	10.2 ± 3.4g	Muscle	30	0.227 ± 0.102	NR	NR	Allard and Stokes 1989
ON: L. Vernon, inlet	1986	Crayfish *Cambarus bartoni*	4.9 ± 3.8 g	Muscle	8	0.304 ± 0.073	NR	NR	Allard and Stokes 1989
ON: Lakes South-central Ontario	1993	Daphnia spp.	Adult >363 µm	Whole	24	NR	0.019 to 0.448 dw	NR	Westcott and Kalff 1996
ON: Little Hawk L.	1986	Crayfish *Cambarus bartoni*	7.1 ± 2.8 g 8.7 ± 3.5 g	Muscle	16	0.073 ± 0.038 0.072 ± 0.020	NR	NR	Allard and Stokes 1989

Location	Year	Species	Life Stage or Size	Tissue	N	Total Hg μg·g^{-1} ww^{a}	MeHg μg·g^{-1} ww^{a}	% MeHg	Reference
ON: Little Hawk L., inlet	1986	Crayfish *Cambarus bartoni*	2.9 ± 1.6 g 2.7 ± 3.1 g	Muscle	6 8	0.197 ± 0.101 0.123 ± 0.020	NR	NR	Allard and Stokes 1989
ON: Opeongo L.	1986	Crayfish *Cambarus bartoni*	4.5 ± 2.2 g	Muscle	31	0.077 ± 0.043	NR	NR	Allard and Stokes 1989
ON: Peninsula L.	1986	Crayfish *Cambarus bartoni*	5.4 ± 1.8 g	Muscle	40	0.069 ± 0.030	NR	NR	Allard and Stokes 1989
ON: Rebecca L.	1986	Crayfish *Cambarus bartoni*	10.0 ± 4.0 g	Muscle	33	0.145 ± 0.070	NR	NR	Allard and Stokes 1989
ON: Rebecca L.	1986	Crayfish *Orconectes virilis*	9.3 ± 6.5 g	Muscle	27	0.059 ± 0.035	NR	NR	Allard and Stokes 1989
ON: Roughrock L.	1980	Crayfish *Orconectes virilis*	Adult	Muscle	11	0.16 ± 0.06	NR	NR	Parks et al. 1991
ON: Round L.	1986	Crayfish *Cambarus bartoni*	4.5 ± 2.5 g	Muscle	35	0.081 ± 0.047	NR	NR	Allard and Stokes 1989
ON: Sand L.	1979	Crayfish *Orconectes virilis*	Adult	Muscle	14	0.26 ± 0.11	NR	NR	Parks et al. 1991
ON: Umfreville L.	1980	Crayfish *Orconectes virilis*	Adult	Muscle	4	0.25 ± 0.08	NR	NR	Parks et al. 1991
ON: Wabigoon L.	1987	Crayfish *Orconectes virilis*	Adult	Muscle	28	0.15^{d}	NR	NR	Parks et al. 1988

Location	Year	Species	Life Stage or Size	Tissue	N	Total Hg µg·g^{-1} ww^{a}	MeHg µg·g^{-1} ww^{a}	% MeHg	Reference
ON: Wabigoon L.	1979	Crayfish *Orconectes virilis*	Adult	Muscle	28	0.16 ± 0.16	NR	NR	Parks et al. 1991
ON: Wabigoon L.	1980	Crayfish *Orconectes virilis*	Adult	Muscle	30	0.09 ± 0.04	NR	NR	Parks et al. 1991
ON: Wabigoon L.	1981	Crayfish *Orconectes virilis*	Adult	Muscle	4	0.07 ± 0.03	NR	NR	Parks et al. 1991
ON: Wabigoon R., inflow to Ball L.	1979	Crayfish *Orconectes virilis*	Adult	Muscle	21	0.80 ± 0.20	NR	NR	Parks et al. 1991
ON: Wabigoon R., inflow to Ball L.	1980	Crayfish *Orconectes virilis*	Adult	Muscle	29	1.7 ± 0.49	NR	NR	Parks et al. 1991
ON: Wabigoon R., inflow to Ball L.	1981	Crayfish *Orconectes virilis*	Adult	Muscle	40	0.91 ± 0.25	NR	NR	Parks et al. 1991
ON: Wabigoon R., inflow to Clay L.	1980	Crayfish *Orconectes virilis*	Adult	Muscle	30	0.79 ± 0.26	NR	NR	Parks et al. 1991
ON: Wabigoon R., inflow to Clay L.	1981	Crayfish *Orconectes virilis*	Adult	Muscle	13	0.76 ± 0.20	NR	NR	Parks et al. 1991
ON: Wabigoon R., outflow of Clay L.	1981	Crayfish *Orconectes virilis*	Adult	Muscle	15	0.75 ± 0.25	NR	NR	Parks et al. 1991
ON: Waseosa L.	1986	Crayfish *Orconectes propinquus*	2.5 ± 1.0 g 8.6 ± 3.9 g 3.4 ± 2.3 g	Muscle	34 23 23	0.095 ± 0.052 0.082 ± 0.034 0.083 ± 0.036	NR	NR	Allard and Stokes 1989

Location	Year	Species	Life Stage or Size	Tissue	N	Total Hg µg·g^{-1} ww^{a}	MeHg µg·g^{-1} ww^{a}	% MeHg	Reference
QC: Duncan L., control for La Grande Reservoir	1992	Coleoptera Dytiscidae	Larvae/ Pupae	Whole	11	0.175 dw	0.107 dw	61%	Tremblay et al. 1996
QC: Duncan L., control for La Grande Reservoir	1992	Corixidae *Sigara* spp.	Larvae/ Pupae	Whole	17	0.256 dw	0.124 dw	49%	Tremblay et al. 1996
QC: Duncan L., control for La Grande Reservoir	1992	Ephemeroptea *Leptophlebia* spp.	Larvae/ Pupae	Whole	11	0.129 dw	0.014 dw	11%	Tremblay et al. 1996
QC: Duncan L., control for La Grande Reservoir	1992	Odonata *Somatochlora* spp.	Larvae/ Pupae	Whole	4	0.136 dw	0.102 dw	75%	Tremblay et al. 1996
QC: Duncan L., control for La Grande Reservoir	1992	Trichoptera *Agrypnia* spp	Larvae/ Pupae	Whole	5	0.124 dw	0.061 dw	50%	Tremblay et al. 1996
QC: Duncan L., control for La Grande Reservoir	1992	Trichoptera *Grammotaulius* spp.	Larvae/ Pupae	Whole	4	0.120 dw	0.056 dw	47%	Tremblay et al. 1996
QC: Duncan L., control for La Grande Reservoir	1992	Trichoptera *Asynarchus* spp	Larvae/ Pupae	Whole	10	0.143 dw	0.029 dw	20%	Tremblay et al. 1996
QC: La Grande-1 Reservoir	1992	Chironomids	Larvae/ Pupae	Whole	12	1.020 dw	0.064 dw	6%	Tremblay et al. 1996
QC: La Grande-1 Reservoir	1992	Corixidae *Sigara* spp.	Larvae/ Pupae	Whole	10	0.514 dw	0.411 dw	80%	Tremblay et al. 1996

Location	Year	Species	Life Stage or Size	Tissue	N	Total Hg $\mu g \cdot g^{-1}$ ww^a	MeHg $\mu g \cdot g^{-1}$ ww^a	% MeHg	Reference
QC: La Grande-2 Reservoir (Dike 14)	1992	Coleoptera Dytiscidae	Larvae/ Pupae	Whole	12	0.621 dw	0.473 dw	76%	Tremblay et al. 1996
QC: La Grande-2 Reservoir (Dike 14)	1992	Corixidae *Sigara* spp.	Larvae/ Pupae	Whole	14	0.551 dw	NR	NR	Tremblay et al. 1996
QC: La Grande-2 Reservoir (Dike 14)	1992	Ephemeroptera *Leptophlebia* spp.	Larvae/ Pupae	Whole	32	0.139 dw	0.069 dw	49%	Tremblay et al. 1996
QC: La Grande-2 Reservoir (Dike 14)	1992	Trichoptera *Agrypnia* spp.	Larvae/ Pupae	Whole	4	0.292 dw	0.059 dw	20%	Tremblay et al. 1996
QC: La Grande-2 Reservoir North arm	1992	Chironomids	Larvae/ Pupae	Whole	8	0.285 dw	0.073 dw	26%	Tremblay et al. 1996
QC: La Grande-2 Reservoir North arm	1992	Corixidae *Sigara* spp.	Larvae/ Pupae	Whole	13	1.371 dw	1.043 dw	76%	Tremblay et al. 1996
QC: La Grande-2 Reservoir North arm	1992	Odonata *Somatochlora* spp.	Larvae/ Pupae	Whole	5	0.283 dw	0.211 dw	75%	Tremblay et al. 1996
QC: La Grande-2 Reservoir North arm	1992	Trichoptera *Asynarchus* spp.	Larvae/ Pupae	Whole	19	0.330 dw	0.081 dw	24%	Tremblay et al. 1996
QC: La Grande-2 Reservoir South arm	1992	Chironomids	Larvae/ Pupae	Whole	66	0.501 dw	0.076 dw	15%	Tremblay et al. 1996
QC: La Grande-2 Reservoir South arm	1992	Corixidae *Sigara* spp.	Larvae/ Pupae	Whole	44	1.675 dw	1.519 dw	91%	Tremblay et al. 1996

Location	Year	Species	Life Stage or Size	Tissue	N	Total Hg µg·g^{-1} ww[a]	MeHg µg·g^{-1} ww[a]	% MeHg	Reference
QC: La Grande-2 Reservoir South arm	1992	Ephemeroptea *Leptophlebia* spp.	Larvae/ Pupae	Whole	7	0.224 dw	0.081 dw	36%	Tremblay et al. 1996
QC: La Grande-2 Reservoir South arm	1992	Odonata *Somatochlora* spp.	Larvae/ Pupae	Whole	3	0.615 dw	0.615 dw	100%	Tremblay et al. 1996
QC: La Grande-2 Reservoir South arm	1992	Trichoptera *Asynarchus* spp.	Larvae/ Pupae	Whole	10	0.544 dw	0.106 dw	20%	Tremblay et al. 1996
SK: Buffalo Pound L.	1985	Freshwater mussel *Pyganodon grandis*[v]	Adult	Muscle	12	0.20 ± 0.07	NR	NR	Hammer et al. 1988

NR = Not Reported

Notes:

[a] Unless noted otherwise, i.e., dw = dry weight.

[b] Non-zooplankton debris was not separated from bulk samples.

[c] Formerly *Anodonta grandis grandis*

[d] Authors assumed the majority of THg to be MeHg.

APPENDIX B-2B: LEVELS OF TOTAL MERCURY AND METHYLMERCURY IN MARINE INVERTEBRATES IN THE CANADIAN ENVIRONMENT.

Location	Year	Species	Life Stage or size	Tissue	N	Total Hg $\mu g \cdot g^{-1}$ ww	MeHg $\mu g \cdot g^{-1}$ ww	% MeHg	Reference
QC: Saguenay Fjord, mouth	1976-1977	Mussel Mytilus edulis	Adult	Soft tissue	NR	0.629 0.165	NR	NR	Cossa and Rondeau 1985
QC: South shore, lower estuary	1976-1977	Mussel *Mytilus edulis*	Adult	Soft tissue	NR	0.459 ± 0.041 0.267 ± 0.034	NR	NR	Cossa and Rondeau 1985
QC: Gaspé Peninsula	1976-1977	Mussel *Mytilus edulis*	Adult	Soft tissue	NR	0.301 ± 0.071 0.120 ± 0.015	NR	NR	Cossa and Rondeau 1985

APPENDIX B-3A: LEVELS OF TOTAL MERCURY AND METHYL MERCURY IN FRESHWATER FISH IN THE CANADIAN ENVIRONMENT.

Location	Year	Species	Life Stage, Age or Size	Sex	Tissue	N	Total Hg $\mu g \cdot g^{-1}$ ww^{a}	MeHg $\mu g \cdot g^{-1}$ ww^{a}	Other information	Reference
AB: Giauque L. 63° 10 N	1977	Lake trout *Salvelinus namaycush*	Adult 10-22 years	NR	Muscle	31	3.79 mean 1.38 to 12.30	NR	Contaminated	Moore and Sutherland 1980
AB: Giauque L. 63° 10 N	1977	Northern pike *Esox lucius*	Adult 8-22 years	NR	Muscle	27	1.75 mean 0.55 to 4.80	NR	Contaminated	Moore and Sutherland 1980
AB: Giauque L. 63° 10 N	1977	Round whitefish *Prosopium cylindraceum*	Adult 9-20 years	NR	Muscle	35	1.22 mean 0.44 to 2.64	NR	Contaminated	Moore and Sutherland 1980
AB: Hidden L., south of Thompson L.	1978	Lake whitefish *Coregonus clupeaformis*	Adult 4-17 years	NR	Muscle	13	0.20 mean 0.02 to 0.39	NR	Control for Thompson L.	Moore and Sutherland 1980
AB: North Saskatchewan R., downstream of Edmonton	1982	Goldeye *Hiodon alosoides*	NR	NR	Muscle	69	0.594 ± 0.074 (95% CI) 0.294 to 1.766	NR	93% MeHg	Ramamoorthy et al. 1985
AB: North Saskatchewan R., downstream of Edmonton	1982	Longnose sucker *Catostomus catostomus*	NR	NR	Muscle	46	0.245 ± 0.029 (95% CI) 0.062 to 0.548	NR	77% MeHg	Ramamoorthy et al. 1985
AB: North Saskatchewan R., downstream of Edmonton	1982	Northern pike *Esox lucius*	NR	NR	Muscle	43	0.314 ± 0.066 (95% CI) 0.044 to 1.039	NR	90% MeHg	Ramamoorthy et al. 1985
AB: North Saskatchewan R., downstream of Edmonton	1982	Northern redhorse sucker *Moxostoma macrolepidotum*	NR	NR	Muscle	23	0.368 ± 0.047 (95% CI) 0.212 to 0.619	NR	87% MeHg	Ramamoorthy et al. 1985

Location	Year	Species	Life Stage, Age or Size	Sex	Tissue	N	Total Hg $\mu g \cdot g^{-1}$ ww^{a}	MeHg $\mu g \cdot g^{-1}$ ww^{a}	Other Information	Reference
AB: N. Saskatchewan R., downstream of Edmonton	1982	Sauger *Stizostedion canadense*	NR	NR	Muscle	26	0.743 ± 0.094 (95% CI) 0.271 to 1.585	NR	92% MeHg	Ramamoorthy et al. 1985
AB: N. Saskatchewan R. downstream of Edmonton	1982	Walleye *Stizostedion vitreum*	NR	NR	Muscle	35	0.645 ± 0.104 (95% CI) 0.140 to 1.202	NR	93% MeHg	Ramamoorthy et al. 1985
AB: N. Saskatchewan R., downstream of Edmonton	1982	White sucker *Catostomus commersoni*	NR	NR	Muscle	27	0.313 ± 0.061 (95% CI) 0.028 to 0.659	NR	87% MeHg	Ramamoorthy et al. 1985
AB: N. Saskatchewan R., upstream of Edmonton	1982	Goldeye *Hiodon alosoides*	NR	NR	Muscle	10	0.452 ± 0.072 (95% CI) 0.295 to 0.633	NR	93% MeHg	Ramamoorthy et al. 1985
AB: N. Saskatchewan R., upstream of Edmonton	1982	Walleye *Stizostedion vitreum*	NR	NR	Muscle	8	0.369 ± 0.112 (95% CI) 0.228 to 0.625	NR	95% MeHg	Ramamoorthy et al. 1985
AB: Thistlethwaite, Unnamed Lakes	1977-1978	Lake trout *Salvelinus namaycush*	Adult 5-19 years	NR	Muscle	32	0.11 to 1.00	NR	Controls	Moore and Sutherland 1980
AB: Thistlethwaite, Unnamed, Hidden Lakes	1977-1978	Northern pike *Esox lucius*	Adult 12-26 years	NR	Muscle	22	0.05 to 1.91	NR	Controls	Moore and Sutherland 1980
AB: Thompson L. 62° 37 N	1978	Lake whitefish *Coregonus clupeaformis*	Adult 3-15 years	NR	Muscle	33	0.20 mean 0.03 to 0.62	NR	Lake Contaminated	Moore and Sutherland 1980
AB: Thompson L. 62° 37 N	1978	Northern pike *Esox lucius*	Adult 5-15 years	NR	Muscle	16	1.69 mean 1.02 to 3.11	NR	Contaminated	Moore and Sutherland 1980

Location	Year	Species	Life Stage, Age or Size	Sex	Tissue	N	Total Hg $\mu g \cdot g^{-1}$ ww[a]	MeHg $\mu g \cdot g^{-1}$ ww[a]	Other information	Reference
BC: Kamloops L.	1995	Burbot *Lota lota*	NR	NR	Liver	5	< 0.05	0.01 0.01 to 0.02	–	McDonald et al. 1999[c]
BC: Lakes (5)	1982-1987	Arctic grayling *Thymallus arcticus*	Adult	NR	Liver	1	0.08	NR	–	Rieberger 1992
BC: Lakes (5)	1982-1987	Arctic grayling *Thymallus arcticus*	Adult	NR	Muscle	21	0.08 ± 0.04 S.D	NR	–	Rieberger 1992
BC: Lakes (11)	1982-1987	Cutthroat trout *Salmo clarki*	Adult	NR	Liver	13	0.35 ± 0.25 S.D.	NR	–	Rieberger 1992
BC: Lakes (11)	1982-1987	Cutthroat trout *Salmo clarki*	Adult	NR	Muscle	54	0.29 ± 0.25 S.D.	NR	–	Rieberger 1992
BC: Lakes (12)	1982-1987	Dolly Varden Char *Salvelinus malma*	Adult	Nr	Liver	9	0.11 ± 0.12 S.D.	NR	–	Rieberger 1992
BC: Lakes (12)	1982-1987	Dolly Varden Char *Salvelinus malma*	Adult	NR	Muscle	46	0.15 ± 0.22 S.D.	NR	–	Rieberger 1992
BC: Lakes (5)	1982-1987	Lake trout *Salvelinus namaycush*	Adult	NR	Liver	3	0.09 ± 0.04 S.D.	NR	–	Rieberger 1992
BC: Lakes (5)	1982-1987	Lake trout *Salvelinus namaycush*	Adult	NR	Muscle	25	0.26 ± 0.26 S.D.	NR	–	Rieberger 1992
BC: Lakes (4)	1982-1987	Mountain whitefish *Prosopium williamsoni*	Adult	NR	Liver	3	0.12 ± 0.02 S.D.	NR	–	Rieberger 1992
BC: Lakes (4)	1982-1987	Mountain whitefish *Prosopium williamsoni*	Adult	NR	Muscle	15	0.11 ± 0.09 S.D.	NR	–	Rieberger 1992

Location	**Year**	**Species**	**Life Stage, Age or Size**	**Sex**	**Tissue**	**N**	**Total Hg µg·g^{-1} ww^{a}**	**MeHg µg·g^{-1} ww^{a}**	**Other information**	**Reference**
BC: Lakes (24)	1982-1987	Rainbow trout *Oncorhynchus mykiss*	Adult	NR	Liver	17	0.11 ± 0.11 S.D.	NR	–	Rieberger 1992
BC: Lakes (24)	1982-1987	Rainbow trout *Oncorhynchus mykiss*	Adult	NR	Muscle	97	0.09 ± 0.05 S.D.	NR	–	Rieberger 1992
BC: Moose L.	1995	Burbot *Lota lota*	NR	NR	Liver	5	0.03 0.02 to 0.08	0.04 < 0.05 to 0.12	–	McDonald et al. 1999^{c}
BC: Nicola L	1995	Burbot *Lota lota*	NR	NR	Liver	5	0.04 < 0.05 to 0.08	0.03 < 0.05 to 0.07	–	McDonald et al. 1999
BC: Northern lakes (5)	1970s	Burbot *Lota lota*	51 – 71 cm	NR	Muscle or Whole	Var	0.25 to 0.46 range of means	NR	Pinchi Fault area	Watson 1992
BC: Northern lakes (3)	1970s	Kokanee *Oncorhynchus nerka kennerlyi*	1000–3500 g	NR	Muscle or Whole	Var	0.03 to 0.07 range of means	NR	Pinchi Fault area	Watson 1992
BC: Northern lakes (14)	1970s 1980s	Lake trout *Salvelinus namaycush*	28 – 99 cm	NR	Muscle or Whole	Var	0.17 to 0.63 range of means	NR	Pinchi Fault area	Watson 1992
BC: Northern lakes (10)	1970s 1980s	Rainbow trout *Oncorhynchus mykiss*	23 – 77 cm	NR	Muscle or Whole	Var	0.10 to 0.27 range of means	NR	Pinchi Fault area	Watson 1992
BC: Northern rivers	1970s 1980s	Bull trout *Salvelinus confluentus*	20 – 85 cm	NR	Muscle or Whole	Var	0.02 to 0.78 range of means	NR	–	Watson 1992
BC: Northern rivers	1970s 1980s	Lake whitefish *Coregonus clupeaformis*	32 – 51 cm	NR	Muscle or Whole	21	0.07 to 0.26 range of means	NR	–	Watson 1992
BC: Northern rivers	1970s 1980s	Rainbow trout *Oncorhynchus mykiss*	15 – 42 cm	NR	Muscle or Whole	37	0.03 to 0.12 range of means	NR	–	Watson 1992

Location	Year	Species	Life Stage, Age or Size	Sex	Tissue	N	Total Hg $\mu g \cdot g^{-1}$ ww^{a}	MeHg $\mu g \cdot g^{-1}$ ww^{a}	Other information	Reference
BC: Pinchi L.	1986	Bull trout *Salvelinus confluentus*	30 – 36 cm	NR	Muscle or Whole	3	0.65 mean 0.49 to 0.75	NR	High natural Hg	Watson 1992
BC: Pinchi L.	1975	Kokanee *Oncorhynchus nerka kennerlyi*	22 – 26 cm	NR	Muscle or Whole	6	0.48 mean 0.32 to 0.76	NR	High natural Hg	Watson 1992
BC: Pinchi L.	1970s 1986	Lake trout *Salvelinus namaycush*	24 – 72 cm	NR	Muscle or Whole	Var	1.06 to 5.78 range of means	NR	High natural Hg	Watson 1992
BC: Pinchi L.	1975-1986	Lake whitefish *Coregonus clupeaformis*	26 – 46 cm	NR	Muscle or Whole	Var	0.65 to 0.90 range of means	NR	High natural Hg	Watson 1992
BC: Pinchi L.	1970s 1986	Rainbow trout *Oncorhynchus mykiss*	14 – 32 cm	NR	Muscle or Whole	16	0.20 to 0.40 range of means	NR	High natural Hg	Watson 1992
BC: Stuart L.	1995	Burbot *Lota lota*	NR	NR	Liver	5	0.04 < 0.05 to 0.10	0.03 0.02 to 0.09	–	McDonald et al. 1999
BC: Williston L., behind Bennett Dam	1980s	Bull trout *Salvelinus confluentus*	20 – 88 cm	NR	Muscle or Whole	Var	0.25 to 1.62 range of site means	NR	–	Watson 1992
BC: Williston L., behind Bennett Dam	1980s	Burbot *Lota lota*	24 – 83 cm	NR	Muscle or Whole	Var	0.26 to 0.33 range of site means	NR	–	Watson 1992
BC: Williston L., behind Bennett Dam	1980s	Kokanee *Oncorhynchus nerka kennerlyi*	20 – 31 cm	NR	Muscle or Whole	Var	0.04 to 0.31 range of site means	NR	–	Watson 1992
BC: Williston L., behind Bennett Dam	1980	Lake trout *Salvelinus namaycush*	39 – 49 cm	NR	Muscle or Whole	Var	0.24 to 0.32 range of site means	NR	–	Watson 1992

Location	Year	Species	Life Stage, Age or Size	Sex	Tissue	N	Total Hg $\mu g \cdot g^{-1}$ ww[a]	MeHg $\mu g \cdot g^{-1}$ ww[a]	Other Information	Reference
BC: Williston L., behind Bennett Dam	1980s	Rainbow trout *Oncorhynchus mykiss*	22 – 41 cm	NR	Muscle or Whole	Var	0.04 to 0.08 range of site means	NR	–	Watson 1992
MB: Cliff L.	1976	Northern pike *Esox lucius*	1-7 years	NR	Liver	23	0.06 - 0.72	NR	Near a base metal smelter	McFarlane and Franzin 1980
MB: Footprint L., downstream of Notigi Reservoir	1978-1981	Northern pike *Esox lucius*	45.7 – 52.6 cm	NR	Muscle	58	0.81 mean of all site means for 3 years.	NR	Churchill River flooding	Bodaly et al. 1984
MB: Footprint L., downstream of Notigi Reservoir	1978-1981	Walleye *Stizostedion vitreum*	37.6 – 39.4 cm	NR	Muscle	82	0.95 mean of all site means for 3 years.	NR	Churchill River flooding	Bodaly et al. 1984
MB: Hamell L.	1976	Northern pike *Esox lucius*	2-5 years	NR	Liver	18	0.08 to 0.86	NR	Near a base metal smelter	McFarlane and Franzin 1980
MB: Isset L., Notigi Reservoir	1975-1982	Lake whitefish *Coregonus clupeaformis*	31.0 – 38.4 cm	NR	Muscle	54	0.23 mean of all site means for 3 years.	NR	Churchill River flooding	Bodaly et al. 1984
MB: Isset L., Notigi Reservoir	1978-1982	Northern pike *Esox lucius*	57.3 – 59.6 cm	NR	Muscle	31	0.76 mean of all site means for 2 years.	NR	Churchill River flooding	Bodaly et al. 1984
MB: Isset L., Notigi Reservoir	1978-1982	Walleye *Stizostedion vitreum*	34.7 – 38.9 cm	NR	Muscle	24	1.16 mean of all site means for 2 years.	NR	Churchill River flooding	Bodaly et al. 1984
MB: Mystery L., downstream of Notigi Reservoir	1979	Northern pike *Esox lucius*	54.1 cm (mean)	NR	Muscle	45	0.79 mean of all site means for 1 year.	NR	Churchill River flooding	Bodaly et al. 1984
MB: Mystery L., downstream of Notigi Reservoir	1979	Walleye *Stizostedion vitreum*	46.2 cm	NR	Muscle	33	1.13	NR	Churchill River flooding	Bodaly et al. 1984

Location	Year	Species	Life Stage, Age or Size	Sex	Tissue	N	Total Hg $\mu g \cdot g^{-1}$ ww^a	MeHg $\mu g \cdot g^{-1}$ ww^a	Other information	Reference
MB: Notigi L., Notigi Reservoir	1980-1981	Lake whitefish *Coregonus clupeaformis*	30.9 – 42.1 cm	NR	Muscle	44	0.18 mean of all site means for 2 years.	NR	Churchill River flooding	Bodaly et al. 1984
MB: Notigi L., Notigi Reservoir	1977-1982	Northern pike *Esox lucius*	58.1 – 78.8 cm	NR	Muscle	68	1.83 mean of all site means for 4 years.	NR	Churchill River flooding	Bodaly et al. 1984
MB: Notigi L., Notigi Reservoir	1978-1982	Walleye *Stizostedion vitreum*	41.6 – 50.3 cm	NR	Muscle	62	1.89 mean of all site means for 4 years.	NR	Churchill River flooding	Bodaly et al. 1984
MB: Rat L., Notigi Reservoir	1978-1980	Lake whitefish *Coregonus clupeaformis*	42.2 – 43.9 cm	NR	Muscle	31	0.36 mean of all site means for 4 years.	NR	Churchill River flooding	Bodaly et al. 1984
MB: Rat L., Notigi Reservoir	1978-1980	Northern pike *Esox lucius*	69.8 cm	NR	Muscle	30	2.17 mean of all site means for 2 years.	NR	Churchill River flooding	Bodaly et al. 1984
MB: Rat L., Notigi Reservoir	1978-1980	Walleye *Stizostedion vitreum*	40.8 – 45.0 cm	NR	Muscle	79	2.14 mean of all site means for 3 years.	NR	Churchill River flooding	Bodaly et al. 1984
MB: Southern Indian L.	1975-1882	Lake whitefish *Coregonus clupeaformis*	31.6 – 42.3 cm	NR	Muscle	700	0.13 mean of all site means for 6 years.	NR	–	Bodaly et al. 1984
MB: Southern Indian L.	1975-1882	Northern pike *Esox lucius*	49.7 – 66.6 cm means	NR	Muscle	461	0.68 mean of all site means for 4-5 years.	NR	–	Bodaly et al. 1984
MB: Southern Indian L.	1975-1882	Walleye *Stizostedion vitreum*	33.3 – 43.5 cm means	NR	Muscle	289	0.57 mean of all site means for 4-5 years.	NR	–	Bodaly et al. 1984
MB: Thompson L.	1976	Northern pike *Esox lucius*	1-6 years.	NR	Liver	22	0.09 to 0.64	NR	Near a base metal smelter	McFarlane and Franzin 1980
MB: Threepoint L., downstream Notigi Res.	1980-1981	Lake whitefish *Coregonus clupeaformis*	33.3 – 42.9 cm means	NR	Muscle	26	0.40 mean of all site means for 2 years.	NR	Churchill River flooding	Bodaly et al. 1984

Location	Year	Species	Life Stage, Age or Size	Sex	Tissue	N	Total Hg µg·g^{-1} ww^{a}	MeHg µg·g^{-1} ww^{a}	Other Information	Reference
MB: Threepoint L., downstream Notigi Reservoir	1980-1981	Northern pike *Esox lucius*	51.7 – 68.8 cm means	NR	Muscle	38	1.31 mean of all site means for 2 years.	NR	Churchill River flooding	Bodaly et al. 1984
MB: Threepoint L., downstream Notigi Reservoir	1980-1981	Walleye *Stizostedion vitreum*	38.1 – 40.3 cm means	NR	Muscle	52	1.27 mean of all site means for 2 years.	NR	Churchill River flooding	Bodaly et al. 1984
MB: Wapisu L., downstream Notigi Reservoir	1977	Northern pike *Esox lucius*	41.1 cm mean	NR	Muscle	41	0.81 mean of all site means for 1 year.	NR	Churchill River flooding	Bodaly et al. 1984
MB: Wapisu L., downstream Notigi Reservoir	1977	Walleye *Stizostedion vitreum*	41.1 cm mean	NR	Muscle	94	1.25 mean of all site means for 1 year.	NR	Churchill River flooding	Bodaly et al. 1984
MB: Wuskwatim L., downstream Notigi Reservoir	1979-1982	Northern pike *Esox lucius*	51.4 – 60.5 cm means	NR	Muscle	108	1.01 mean of all site means for 4 years.	NR	Churchill River flooding	Bodaly et al. 1984
MB: Wuskwatim L., downstream Notigi Reservoir	1979-1981	Walleye *Stizostedion vitreum*	39.9 – 41.5 cm means	NR	Muscle	143	0.88 mean of all site means for 3 years.	NR	Churchill River flooding	Bodaly et al. 1984
NB: St. John R., Green River Reservoir	1983	Lake whitefish *Coregonus clupeaformis*	20 - 25 cm	NR	Muscle	12	0.12 mean 0.10 to 0.64	NR	–	Bailey 1985
NB. St. John R., Sisson Branch Reservoir	1983	Land-locked salmon *Salmo salar*	38 & 58 cm	NR	Muscle	2	0.84 and 1.5	NR	–	Bailey 1985

Location	Year	Species	Life Stage, Age or Size	Sex	Tissue	N	Total Hg $\mu g \cdot g^{-1}$ ww[a]	MeHg $\mu g \cdot g^{-1}$ ww[a]	Other information	Reference
NF: Labrador	1977-1978	Brook trout *Salvelinus fontinalis*	NR	NR	NR	96	0.01-1.69	NR	--	Bruce et al. 1979
NF: Labrador	1977-1978	Northern pike *Esox lucius*	NR	NR	NR	117	0.01-3.08	NR	--	Bruce et al. 1979
NF: Labrador	1977-1978	Lake trout *Salvelinus namaycush*	NR	NR	NR	107	0.12-3.37	NR	--	Bruce et al. 1979
NF: Labrador Smallwood Reservoir	1977-1978	Land-locked salmon *Salmo salar*	NR	NR	Muscle	NR	1.5 to 2.3	NR	--	Bruce and Spencer 1978 c.f. Bailey 1985
NS: Annapolis R.	NR	Striped bass[d] *Morone saxatilis*	7.38 kg mean	NR	Muscle	NR	0.77 mean 0.26 to 1.97	NR	--	Ray et al. 1984
NS: Wreck Cove Reservoir	1994	Brook trout *Salvelinus fontinalis*	49-211 g 16.8-26.8 cm	NR	Muscle	12	0.495 ± 0.164 0.27 to 0.73	NR	Oldest fish only 3 years	ADI Nolan Davis Inc. 1994
NS: Shubenacadie R.	NR	Striped bass *Morone saxatilis*	1.81 kg mean	NR	Muscle	NR	0.40 0.16 to 1.44	NR	--	Ray et al. 1984
NWT: Hay R.	1989-1990	Lake whitefish *Coregonus clupeaformi*	362 – 1800g 7 – 12 years	7M 33F	Muscle	40	0.07 mean 0.03 to 0.13	NR	--	Grey et al. 1995
NWT: Hay R.	1989-1990	Northern pike *Esox lucius*	775 – 3100g 5 – 13 years	16M 5F	Muscle	21	0.32 mean 0.19 to 0.59	NR	--	Grey et al. 1995
NWT: Hay R.	1989-1990	Walleye *Stizostedion vitreum*	450 – 1800g 4 – 11 years	5M 30F	Muscle	35	0.22 mean 0.1 to 0.32	NR	--	Grey et al. 1995
NWT: Johnny Hoe R./ Keller L.	1993	Lake trout *Salvelinus namaycush*	8-32 years	NR	Muscle	15	0.41 mean 0.22 to 1.05	NR	--	Stephens 1995

Location	Year	Species	Life Stage, Age or Size	Sex	Tissue	N	Total Hg $\mu g \cdot g^{-1}$ ww^{a}	MeHg $\mu g \cdot g^{-1}$ ww^{a}	Other information	Reference
NWT: Johnny Hoe R./ Keller L.	1993	Lake whitefish *Coregonus clupeaformis*	7-19 years	NR	Muscle	15	0.064	NR	--	Stephens 1995
NWT: Johnny Hoe R./ Lac Ste. Thérèse	1993	Lake trout *Salvelinus namaycush*	32-39 years	NR	Muscle	2	1.34	NR	Unknown source	Stephens 1995
NWT: Johnny Hoe R./ Lac Ste. Thérèse	1993	Northern pike *Esox lucius*	6-11 years	NR	Muscle	4	0.735 mean 0.25 to 1.09	NR	Unknown source	Stephens 1995
NWT: Johnny Hoe R./ Lac Ste. Thérèse	1993	Walleye *Stizostedion vitreum*	9-17 years	NR	Muscle	30	1.49 mean 0.29 to 1.99	NR	Unknown source	Stephens 1995
NWT: Johnny Hoe R./ Tseepantee L.	1993	Lake whitefish *Coregonus clupeaformis*	6-21 years	NR	Muscle	15	0.102	NR	--	Stephens 1995
NWT: Johnny Hoe R./ Tseepantee L.	1993	Northern pike *Esox lucius*	6-8 years	NR	Muscle	6	0.475 mean 0.39 to 0.71	NR	--	Stephens 1995
NWT: Johnny Hoe R/ Tseepantee L.	1993	Walleye *Stizostedion vitreum*	11-23 years	NR	Muscle	15	0.926 mean 0.25 to 1.42	NR	--	Stephens 1995
NWT: Kaminak L., West of Hudson Bay	1970-1971	Lake trout *Salvelinus namaycush*	Adult	NR	Muscle or Whole	70	0.57 to 2.0	NR	High natural Hg	Shilts and Coker 1995
NWT: Leland L.	1989-1990	Lake whitefish *Coregonus clupeaformis*	800 – 1600 g 7 – 12 years	10M 10F	Muscle	20	0.11 mean 0.05 to 0.23	NR	--	Grey et al. 1995
NWT: Leland L.	1989-1990	Northern pike *Esox lucius*	500 – 2880 g 4 – 14 years	14M 19F	Muscle	32	0.34 mean 0.16 to 0.59	NR	--	Grey et al. 1995

Location	Year	Species	Life Stage, Age or Size	Sex	Tissue	N	Total Hg $\mu g \cdot g^{-1}$ ww^a	MeHg $\mu g \cdot g^{-1}$ ww^a	Other information	Reference
NWT: Leland L.	1989-1990	Walleye *Stizostedion vitreum*	776 – 2450 g 8 – 18 years	11M 8F	Muscle	19	0.46 mean 0.25 to 0.73	NR	--	Grey et al. 1995
NWT: Slave R.	1988-1990	Lake whitefish *Coregonus clupeaformis*	524 – 1025 g 6 – 12 years	17M 13F	Muscle	30	0.08 mean 0.02 to 0.13	NR	--	Grey et al. 1995
NWT: Slave R.	1988-1990	Northern pike *Esox lucius*	700 – 2750 g 5 – 15 years	24M 39F	Muscle	63	0.34 mean 0.17 to 0.60	NR	--	Grey et al. 1995
NWT: Slave R.	1988-1990	Walleye *Stizostedion vitreum*	500 – 1600 g 6 – 14 years	72M 27F	Muscle	99	0.34 mean 0.13 to 0.80	NR	--	Grey et al. 1995
ON: Ball L. North-west Ontario	1970	Northern pike *Esox lucius*	1529 – 2900 g	NR	Muscle	7	7.7 mean 1.61 to 20.0	NR	Contaminated	Fimreite and Reynolds 1973
ON: Ball L. North-west Ontario	1970	Rock bass *Ambloplites rupestris*	650 – 1685 g	NR	Muscle	9	6.2 mean 1.14 to 10.9	NR	Contaminated	Fimreite and Reynolds 1973
ON: Ball L. North-west Ontario	1970	Walleye *Stizostedion vitreum*	410 – 1550 g	NR	Muscle	9	4.5 mean 0.5 to 8.7	NR	Contaminated	Fimreite and Reynolds 1973
ON: Clay L. North-west Ontario	1970	Burbot *Lota lota*	1355 – 2150 g	NR	Muscle	4	21.9 mean 19.1 to 24.8	NR	Contaminated	Fimreite and Reynolds 1973
ON: Clay L. North-west Ontario	1970	Walleye *Stizostedion vitreum*	1025 – 2040 g	NR	Muscle	5	15.7 mean 12.3 to 19.6	NR	Contaminated	Fimreite and Reynolds 1973
ON: Clay L. North-west Ontario	1970	White sucker *Catostomus commersoni*	575 – 1050 g	NR	Muscle	5	3.13 mean 2.3 to 3.8	NR	Contaminated	Fimreite and Reynolds 1973

Location	Year	Species	Life Stage, Age or Size	Sex	Tissue	N	Total Hg µg·g^{-1} ww^{a}	MeHg µg·g^{-1} ww^{a}	Other information	Reference
ON: L. Ontario	1977-1988	Lake trout *Salvelinus namaycush*	1 – 8 years	NR	Whole	134	0.20 mean of all sites 0.18 to 0.23	NR	[Hg] increased with weight	Borgmann and Whittle 1991
ON: L. Simcoe	1982	Northern pike *Esox lucius*	1 - 11 years	NR	Muscle	43	Max of 0.85	NR	–	Mathers and Johansen 1985
ON: L. Simcoe	1982	Walleye *Stizostedion vitreum*	1 - 16 years	NR	Muscle	46	Max of 2.7	NR	–	Mathers and Johansen 1985
ON: Lakes	NR	Lake trout *Salvelinus namaycush*	44 cm	NR	Muscle	41	0.303 mean 0.070 to 1.033	NR	–	Wren 1991
ON: Lakes (3) Wabigoon/English R. area, North-west Ontario	1970	Lake trout *Salvelinus namaycush*	1195 – 2400 g	NR	Muscle	13	0.52 to 0.82 range of means all lakes	NR	Control lakes	Fimreite and Reynolds 1973
ON: Lakes (28) from 44°53' to 49°37' Lat.	1975-1984	Lake trout *Salvelinus namaycush*	1213 g (geom. mean)	NR	Whole or muscle	569	0.47 mean of all lake means	NR	With pelagic forage fish, no *Mysis*	Cabana et al. 1994
ON: Lakes (33) from 44°53' to 49°37' Lat.	1975-1984	Lake trout *Salvelinus namaycush*	1615 g (geom. mean)	NR	Whole or muscle	851	0.65 mean of all lake means	NR	With pelagic forage fish and *Mysis*	Cabana et al. 1994
ON: Lakes (35) from 45°01' to 49°10' Lat.	1975-1984	Lake trout *Salvelinus namaycush*	686 g (geom. mean)	NR	whole or muscle	885	0.18 mean of all lake means	NR	With *Mysis*, no pelagic forage fish	Cabana et al. 1994
ON: Lakes Koshlong, Rosseau, Tadenac	1977-1981	Lake trout *Salvelinus namaycush*	60 cm	NR	Muscle	84	0.61 to 0.99 means	NR	Rated moderate [Hg]	MacCrimmon et al. 1983
ON: Lakes Muskoka, Mary, Vernon	1977-1981	Lake trout *Salvelinus namaycush*	60 cm	NR	Muscle	84	2.96 to 4.49 means	NR	Rated high [Hg]	MacCrimmon et al. 1983

Location	Year	Species	Life Stage, Age or Size	Sex	Tissue	N	Total Hg µg·g^{-1} ww[a]	MeHg µg·g^{-1} ww[a]	Other information	Reference
ON: Lakes Simcoe, Joseph, Bella	1977-1981	Lake trout *Salvelinus namaycush*	60 cm	NR	Muscle	85	0.39 to 0.55 means	NR	Rated low [Hg]	MacCrimmon et al. 1983
ON: Lakes (7) Wabigoon/English R. area, North-west Ontario	1970	Northern pike *Esox lucius*	580 – 4370 g	NR	Muscle	27	0.52 to 1.3 range of means all lakes	NR	Control lakes	Fimreite and Reynolds 1973
ON: Lakes (170) Province-wide	1978-1984	Northern pike *Esox lucius*	Adult ~54 cm	NR	Muscle	10+ PL[e]	0.50 mean 0.07 to 1.28	NR	–	Wren et al. 1991
ON: Lakes (16) South-central Ontario	1981	Pumpkinseed sunfish *Lepomis gibbosus*	Adult	NR	Muscle	20[f]	0.01 to 0.54	NR	–	Wren and MacCrimmon 1983
ON: Lakes Koshlong, Rosseau, Tadenac	1977-1981	Rainbow smelt *Osmerus mordax*	15 cm	NR	Muscle	22	0.18 to 0.26 means	NR	Rated moderate [Hg][g]	MacCrimmon et al. 1983
ON: Lakes Muskoka, Mary, Vernon	1977-1981	Rainbow smelt *Osmerus mordax*	15 cm	NR	Muscle	20	0.34 to 0.49 means	NR	Rated high [Hg][g]	MacCrimmon et al. 1983
ON: Lakes Simcoe, Joseph, Bella	1977-1981	Rainbow smelt *Osmerus mordax*	15 cm	NR	Muscle	30	0.08 to 0.14 means	NR	Rated low [Hg]	MacCrimmon et al. 1983
ON: Lakes	NR	Smallmouth bass *Micropterus dolomieu*	31 cm	NR	Muscle	NR	0.402 mean 0.132 to 0.943	NR	–	Wren 1991
ON: Lakes (9) from 44°55' to 45°24' Lat.	1975-1984	Smallmouth bass *Micropterus dolomieu*	305 g (geom. mean)	NR	NR Whole or muscle	179	0.42 mean of all lake means	NR	No pelagic forage fish or *Mysis*	Cabana et al. 1994

Location	Year	Species	Life Stage, Age or Size	Sex	Tissue	N	Total Hg $\mu g \cdot g^{-1}$ ww^a	MeHg $\mu g \cdot g^{-1}$ ww^a	Other Information	Reference
ON: Lakes (17) from 44°25' to 47°58' Lat.	1975-1984	Smallmouth bass *Micropterus dolomieu*	508 g (geom. mean)	NR	NR Whole or muscle	337	0.43 mean of all lake means	NR	With pelagic forage fish, no *Mysis*	Cabana et al. 1994
ON: Lakes (30) from 44°50' to 50°02' Lat.	1975-1984	Smallmouth bass *Micropterus dolomieu*	489 g (geom. mean)	NR	NR Whole or muscle	605	0.53 mean of all lake means	NR	With pelagic forage fish and *Mysis*	Cabana et al. 1994
ON: Lakes	NR	Walleye *Stizostedion vitreum*	41 cm	NR	Muscle	44	0.517 mean 0.128 to 2.216	NR	–	Wren 1991
ON: Lakes (9) Wabigoon/English R. area, North-west Ontario	1970	Walleye *Stizostedion vitreum*	360 – 2420 g	NR	Muscle	49	0.31 to 0.76 range of means all lakes	NR	Control lakes	Fimreite and Reynolds 1973
ON: Lakes (255) province-wide	1978-1984	Walleye *Stizostedion vitreum*	Adult ~41 cm	NR	Muscle	10+ PL	0.58 mean 0.09 to 3.24	NR	–	Wren et al. 1991
ON: Lakes(6) North-west Ontario- remote	1987-1989	Yellow Perch *Perca flavescens*	Age 1+ 7 – 8.1 cm	NR	Muscle	246	0.04 to 0.14	NR	Inverse relation with lake size	Bodaly et al. 1993
ON: Lakes (16) Muskoka-Haliburton area	1978-1987	Yellow perch *Perca flavescens*	Yearling	NR	Whole	<11 PL	0.031 to 0.233 range of lake means	NR	–	Suns and Hitchin 1990
ON: Ottawa R.	1974	Yellow Perch *Perca flavescens*	Yearling	NR	Whole	32	0.13 to 0.17	NR	68 to 74% organic Hg	Rodgers and Qadri 1982
ON: Ottawa R.	1975	Yellow Perch *Perca flavescens*	Yearling	NR	Whole	16	0.13 ± 0.005	NR	83% organic Hg	Rodgers and Qadri 1982

Location	Year	Species	Life Stage, Age or Size	Sex	Tissue	N	Total Hg μg·g^{-1} ww^{a}	MeHg μg·g^{-1} ww^{a}	Other information	Reference
ON: Tide-Indian-Grassy Narrows L. North-west Ontario	1970	Walleye *Stizostedion vitreum*	715 – 4220 g	NR	Muscle	18	2.7 to 3.4 range of means of 3 lakes	NR	Contaminated	Fimreite and Reynolds 1973
ON: Wabigoon R. North-west Ontario	1970	Northern pike *Esox lucius*	510 – 2700 g	NR	Muscle	3	15.2 mean 8.6 to 27.8	NR	Contaminated	Fimreite and Reynolds 1973
ON: Wabigoon R. North-west Ontario	1970	Walleye *Stizostedion vitreum*	160 – 1490 g	NR	Muscle	4	6.8 mean 0.5 to 10.4	NR	Contaminated	Fimreite and Reynolds 1973
ON: Wabigoon R. North-west Ontario	1970	White sucker *Catostomus commersoni*	570 – 1060 g	NR	Muscle	3	4.2 mean 0.6 to 8.9	NR	Contaminated	Fimreite and Reynolds 1973
QC: Great Whale area, Northern Québec	1989-1990	Northern pike *Esox lucius*	NR	NR	Muscle	75	0.63 ± 0.46	NR	–	Langlois et al. 1995
QC: Great Whale area, Northern. Québec	1989-1990	Lake trout *Salvelinus namaycush*	NR	NR	Muscle	504	0.77 ± 0.66	NR	–	Langlois et al. 1995
QC: La Grande- 2 Reservoir	1982-1988	Lake whitefish *Coregonus clupeaformis*	40 cm	NR	Muscle	NR	0.48 to 0.57 – High value after 5 years.	NR	Flooded 1978/9	Verdon et al. 1991
QC: La Grande- 2 Reservoir	1982-1988	Longnose sucker *Catostomus catostomus*	40 cm	NR	Muscle	NR	0.41 to 0.67 – High value after 5 years.	NR	Flooded 1978/9	Verdon et al. 1991
QC: La Grande -2 Reservoir	1982-1988	Northern pike *Esox lucius*	70 cm	NR	Muscle	NR	1.31 to 2.99 – High value after 9 years.	NR	Flooded 1978/9	Verdon et al. 1991

Location	Year	Species	Life Stage, Age or Size	Sex	Tissue	N	Total Hg $\mu g \cdot g^{-1}$ ww^a	MeHg $\mu g \cdot g^{-1}$ ww^a	Other information	Reference
QC: La Grande- 2 Reservoir	1982-1988	Walleye *Stizostedion vitreum*	40 cm	NR	Muscle	NR	1.92 to 2.80 – High level after 9 years.	NR	Flooded 1978/9	Verdon et al. 1991
QC: Lakes (29), near La Grande complex	1978-1988	Lake whitefish *Coregonus clupeaformis*	40 cm	NR	Muscle	NR	0.07 to 0.30	NR	Control	Verdon et al. 1991
QC: Lakes (29), near La Grande complex	1978-1988	Longnose sucker *Catostomus catostomus*	40 cm	NR	Muscle	NR	0.06 to 0.32	NR	Control	Verdon et al. 1991
QC: Lakes (29), near La Grande complex	1978-1988	Northern pike *Esox lucius*	70 cm	NR	Muscle	NR	0.25 to 0.90	NR	Control	Verdon et al. 1991
QC: Lakes (29), near La Grande complex	1978-1988	Walleye *Stizostedion vitreum*	40 cm	NR	Muscle	NR	0.32 to 1.26	NR	Control	Verdon et al. 1991
QC: Lakes (235) and Rivers (214) across QC	1995	Northern pike *Esox lucius*	40-50 cm 55-70 cm >70 cm	NR	NR	NR	0.41 0.66 1.12	NR	Info from I. Guay 1999, MEF, pers. com.	Laliberté 1996
QC: Lakes (235) and Rivers (214) across QC	1995	Walleye *Stizostedion vitreum*	30-40 cm 40-50 cm >50 cm	NR	NR	NR	0.53 0.78 1.26	NR	Info from I. Guay 1999, MEF, pers. com.	Laliberté 1996
QC: Nottaway-Broadback-Rupert area, South-east coast James Bay	1990-1991	Northern pike *Esox lucius*	NR	NR	Muscle	636	0.93 ± 0.51	NR	–	Langlois et al. 1995

Location	Year	Species	Life Stage, Age or Size	Sex	Tissue	N	Total Hg $\mu g \cdot g^{-1}$ ww[a]	MeHg $\mu g \cdot g^{-1}$ ww[a]	Other information	Reference
QC: Nottaway-Broadback-Rupert area, South-east coast James Bay	1990-1991	Walleye *Stizostedion vitreum*	NR	NR	Muscle	789	0.78 ± 0.46	NR	–	Langlois et al. 1995

NR = Not Reported

Var = Variation in the number of samples

Notes:

[a] Unless noted specifically, most mercury in fish is assumed to be MeHg.

[b] Lake had Hg-laden mine tailings discharged to it; this species had low [Hg] compared with Northern pike.

[c] Data unpublished.

[d] Anadromous on Atlantic coast. Introduced on Pacific coast (Eddy and Underhill 1984).

[e] PL = per lake.

[f] Number of fish per lake, except Georgian Bay where 5 fish were obtained.

[g] Hg rating is for lake trout.

APPENDIX B-3B: LEVELS OF TOTAL MERCURY IN MARINE FISH IN THE CANADIAN ENVIRONMENT.

Location	Year	Species	Life Stage, Age or Size	Sex	Tissue	N	Total Hg $\mu g \cdot g^{-1}$ ww[a]	Other information	Reference
NB: Bay of Fundy, SW	1981	Atlantic herring *Clupea harengus harengus*	1-2 years	NR	Muscle	61	0.005 ± 0.001	–	Braune 1987a
NB: Bay of Fundy, SW	1981	Atlantic herring *Clupea harengus harengus*	3-5 years	NR	Muscle	54	0.008 to 0.015 range of year class means	Hg increased with age	Braune 1987a
NB: Bay of Fundy, SW	1981	Atlantic herring *Clupea harengus harengus*	1-2 years	NR	Whole	76	0.005 to 0.006 range of year class means	–	Braune 1987a
NB: Bay of Fundy, SW	1981	Atlantic herring *Clupea harengus harengus*	3-5 years	NR	Whole	54	0.007 to 0.015 range of year class means	[Hg] ~same as muscle only	Braune 1987a
NS: Medway R., at Greenfield	1972	American eel *Anuilla rostrata*	NR	NR	Muscle	23	0.72 THg, 0.40 MeHg	MeHg 50% on average	Freeman and Horne 1972
QC: coastal James Bay	1987-1994	Greenland cod *Gadus ogac*	400 mm	NR	NR	NR	0.14-0.42	Standardized length	Hydro-Quebéc and Groupe-conseil Génivar Inc. (1997)
QC: coastal James Bay	1987-1994	Fourhorn sculpin *Myoxocephalus quadricornis*	250 mm	NR	NR	NR	0.10-0.55	Standardized length	Hydro-Quebéc and Groupe-conseil Génivar Inc. (1997)

Notes:

[a] Unless noted specifically, most mercury in fish is assumed to be MeHg.

APPENDIX B-4:LEVELS OF TOTAL MERCURY AND METHYLMERCURY IN AMPHIBIANS AND REPTILES IN THE CANADIAN ENVIRONMENT.

Location	Year	Species	Life Stage	Tissue	N	Total Hg µg·g^{-1} ww	MeHg µg·g^{-1} ww	% MeHg	Reference
ON: Gray's Cr., Cornwall area	1990	Snapping Turtle (Reptile) *Chelydra serpentina*	Eggs	Whole	5	0.110	NR	NR	Bonin et al. 1995
ON: Raquette R., Cornwall-Massena area	1990	Snapping Turtle (Reptile) *Chelydra serpentina*	Eggs	Whole	5	0.140	NR	NR	Bonin et al. 1995
ON: St. Lawrence R., Dundee area	1989	Snapping Turtle (Reptile) *Chelydra serpentina*	Eggs	Whole	7	0.090	NR	NR	Bonin et al. 1995
QC: St. Lawrence R., Beauharnois	1990	Snapping Turtle (Reptile) *Chelydra serpentina*	Eggs	Whole	3	0.150	NR	NR	Bonin et al. 1995
QC: Ottawa R., Rigaud	1992	Mudpuppy (Amphibia) *Necturus maculosus*	Adult females	Whole no gonads	6	0.087 ± 0.011 max 0.104	NR	NR	Bonin et al. 1995
QC: Ottawa R., Rigaud	1992	Mudpuppy (Amphibia) *Necturus maculosus*	Adult females	Gonads	6	0.068	NR	NR	Bonin et al. 1995
QC: St. Lawrence R., Beauharnois	1992	Mudpuppy (Amphibia) *Necturus maculosus*	Adult females	Whole no gonads	6	0.239 ± 0.137 max 0.445	NR	NR	Bonin et al. 1995

Location	Year	Species	Life Stage	Tissue	N	Total Hg $\mu g \cdot g^{-1}$ ww	MeHg $\mu g \cdot g^{-1}$ ww	% MeHg	Reference
QC: St. Lawrence R., Beauharnois	1992	Mudpuppy (Amphibia) *Necturus maculosus*	Adult females	Gonads	6	0.193	NR	NR	Bonin et al. 1995
QC: Ottawa R., Vaudreuil	1992	Mudpuppy (Amphibia) *Necturus maculosus*	Adult	Whole	3	0.110	NR	NR	Bonin et al. 1995
QC: Ottawa R./ St. Lawrence R. Montreal East	1992	Mudpuppy (Amphibia) *Necturus maculosus*	Adult	Whole	2	0.110	NR	NR	Bonin et al. 1995
QC: Ottawa R. /St. Lawrence. R., Illes de Sorel	1992	Mudpuppy (Amphibia) *Necturus maculosus*	Adult	Whole	6	0.110	NR	NR	Bonin et al. 1995
QC: St. Lawrence R., Pte. Leblanc	1992	Mudpuppy (Amphibia) *Necturus maculosus*	Adult	Whole	4	0.130	NR	NR	Bonin et al. 1995
QC: St. Lawrence R., Port Lewis	1992	Mudpuppy (Amphibia) *Necturus maculosus*	Adult	Whole	2	0.100	NR	NR	Bonin et al. 1995
QC: St. Lawrence R., Nicolet	1992	Mudpuppy (Amphibia) *Necturus maculosus*	Adult	Whole	3	0.100	NR	NR	Bonin et al. 1995
QC: St. Lawrence R., Ste Anne	1992	Mudpuppy (Amphibia) *Necturus maculosus*	Adult	Whole	14	0.290	NR	NR	Bonin et al. 1995

NR = Not Reported

APPENDIX B-5: LEVELS OF TOTAL MERCURY AND METHYLMERCURY IN FRESHWATER MAMMALS IN THE CANADIAN ENVIRONMENT.

Location	Year	Species	Life Stage	Tissue	N	Total Hg $\mu g \cdot g^{-1}$ ww	MeHg $\mu g \cdot g^{-1}$ ww	% MeHg	Reference
MB: Burntwood R. North-central Manitoba	1979 - 1981	Mink *Mustela vison*	<1 – 6 years 88% <2 years	Brain Kidney Liver	21	0.80 1.76 2.35	NR	NR	Kucera 1983
MB: Duck Mountain, near SK border	1979 - 1981	Mink *Mustela vison*	<1 – 6 years 88% <2 years	Brain Kidney Liver	43	0.19 0.67 0.82	NR	NR	Kucera 1983
MB: Wekusko L. North-central Manitoba	1979 - 1981	Mink *Mustela vison*	<1 – 6 years 88% <2 years	Brain Kidney Liver	19	0.39 1.04 1.46	NR	NR	Kucera 1983
MB: Wekusko L. North-central Manitoba	1979 - 1981	Otter *Lutra canadensis*	<1 – 6 years 75% <2 years	Brain Kidney Liver	17	0.28 1.07 1.75	NR	NR	Kucera 1983
MB: Whiteshell R., South-east Manitoba	1979 - 1981	Mink *Mustela vison*	<1 – 6 years 88% <2 years	Brain Kidney Liver	27	0.42 1.47 1.71	NR	NR	Kucera 1983
MB: Whiteshell R., South-east Manitoba	1979 - 1981	Otter *Lutra canadensis*	<1 – 6 years 75% <2 years	Brain Kidney Liver	6	0.38 1.66 2.27	NR	NR	Kucera 1983
MB: Winnipeg R.	1979 - 1981	Mink *Mustela vison*	<1 – 6 years 88% <2 years	Brain Kidney Liver	62	0.97 2.98 3.93	NR	NR	Kucera 1983

Location	Year	Species	Life Stage	Tissue	N	Total Hg µg·g^{-1} ww	MeHg µg·g^{-1} ww	% MeHg	Reference
MB: Winnipeg R.	1979 - 1981	Otter *Lutra canadensis*	<1 – 6 years 75% <2 years	Brain Kidney Liver	13	0.91 2.37 3.67	NR	NR	Kucera 1983
ON: English R. North-west Ontario	1983 - 1985	Mink *Mustela vison*	NR	Brain Kidney Liver Muscle	11	0.45 to 0.64 0.48 to 5.54 0.56 to 6.9 0.16 to 1.62	NR	NR	Wren et al. 1986
ON: English R. North-west Ontario	1983 - 1985	Otter *Lutra canadensis*	NR	Brain Kidney Liver Muscle	20	0.23 to 7.15 0.58 to 12.6 0.71 to 17.4 0.21 to 12.6	NR	NR	Wren et al. 1986
ON: English R. Muskoka and Sudbury	1983 - 1985	Mink *Mustela vison*	NR	Kidney Liver	35 38	NR	NR	43 53	Wren et al. 1986
ON: English R. Muskoka and Sudbury	1983 - 1985	Otter *Lutra canadensis*	NR	Kidney	32	NR	NR	19	Wren et al. 1986
ON: English R. Muskoka and Sudbury	1983 - 1985	Otter *Lutra canadensis*	NR	Muscle	10	NR	NR	90	Wren et al. 1986
ON: Cambridge	1983 - 1985	Mink *Mustela vison*	NR	Liver	9	0.14 ± 0.08 ND to 0.28	NR	NR	Wren et al. 1986
ON: Herschel Twp. South-central Ontario	1990s	Otter *Lutra* canadensis	<1 – 12 years	Kidney Liver	19	1.67 ±1.09 1.54 ± 0.72	NR	NR	Evans 1995
ON: Muskoka lakes	1983 - 1985	Mink *Mustela vison*	NR	Brain	4	0.55 ± 0.10 0.46 to 0.66	NR	NR	Wren et al. 1986

Location	Year	Species	Life Stage	Tissue	N	Total Hg µg·g^{-1} ww	MeHg µg·g^{-1} ww	% MeHg	Reference
ON: Muskoka lakes	1983 - 1985	Mink *Mustela vison*	NR	Kidney	23	2.49 ± 0.94 0.41 to 4.49	NR	NR	Wren et al. 1986
ON: Muskoka lakes	1983 - 1985	Mink *Mustela vison*	NR	Liver	24	2.17 ± 1.24 0.40 to 6.25	NR	NR	Wren et al. 1986
ON: Muskoka lakes	1983 - 1985	Mink *Mustela vison*	NR	Muscle	11	1.62 ±1.07 0.01 to 4.08	NR	NR	Wren et al. 1986
ON: Muskoka lakes	1983 - 1985	Otter *Lutra canadensis*	NR	Brain	2	0.52 ± 0.18 0.40 to 0.63	NR	NR	Wren et al. 1986
ON: Muskoka lakes	1983 - 1985	Otter *Lutra canadensis*	NR	Kidney	11	1.28 ± 0.72 0.34 to 2.64	NR	NR	Wren et al. 1986
ON: Muskoka lakes	1983 - 1985	Otter *Lutra canadensis*	NR	Liver	12	1.50 ± 0.75 0.20 to 2.46	NR	NR	Wren et al. 1986
ON: Muskoka lakes	1983 - 1985	Otter *Lutra canadensis*	NR	Muscle	6	0.55 ± 0.32 0.25 to 1.21	NR	NR	Wren et al. 1986
ON: Near Parry Sound	NR	Beaver *Castor canadensis*	NR	Kidney Liver Muscle	NR	0.03 0.03 0.03	NR	NR	Wren 1984
ON: Near Parry Sound	NR	Otter *Lutra canadensis*	NR	Intestine Kidney Liver Muscle	NR	0.4 ± 0.2 1.4 ± 0.3 3.0 ± 2.2 0.9 ± 0.2	NR	NR	Wren 1984

Location	Year	Species	Life Stage	Tissue	N	Total Hg $\mu g \cdot g^{-1}$ ww	MeHg $\mu g \cdot g^{-1}$ ww	% MeHg	Reference
ON: Near Parry Sound	NR	Racoon *Procyon lotor*	NR	Kidney Liver Muscle	NR	1.1 ± 0.4 4.5 ± 3.5 0.3 ± 0.1	NR	NR	Wren 1984
ON: Sudbury	1983 - 1985	Mink *Mustela vison*	NR	Brain	2	0.36 ± 0.11 0.28 to 0.44	NR	NR	Wren et al. 1986
ON: Sudbury	1983 - 1985	Mink *Mustela vison*	NR	Liver	31	0.56 ± 0.40 0.07 to 1.75	NR	NR	Wren et al. 1986
ON: Sudbury	1983 - 1985	Mink *Mustela vison*	NR	Muscle	19	0.38 ±0.26 ND to 0.88	NR	NR	Wren et al. 1986
ON: Sudbury	1983 - 1985	Mink *Mustela vison*	NR	Kidney	21	0.59 ± 0.23 0.13 to 1.00	NR	NR	Wren et al. 1986
ON: Sudbury	1983 - 1985	Otter *Lutra canadensis*	NR	Brain	3	0.24 0.23 to 0.25	NR	NR	Wren et al. 1986
ON: Sudbury	1983 - 1985	Otter *Lutra canadensis*	NR	Kidney	23	0.59 ± 0.26 0.05 to 1.31	NR	NR	Wren et al. 1986
ON: Sudbury	1983 - 1985	Otter *Lutra canadensis*	NR	Liver	36	0.86 ± 0.92 ND to 4.14	NR	NR	Wren et al. 1986
ON: Sudbury	1983 - 1985	Otter *Lutra canadensis*	NR	Muscle	17	0.30 ± 0.17 0.07 to 0.17	NR	NR	Wren et al. 1986
ON: Turkey L., Sault Ste. Marie area	1983 - 1985	Mink *Mustela vison*	NR	Liver	16	2.36 ± 1.63 1.12 to 7.50	NR	NR	Wren et al. 1986

Location	Year	Species	Life Stage	Tissue	N	Total Hg $\mu g \cdot g^{-1}$ ww	MeHg $\mu g \cdot g^{-1}$ ww	% MeHg	Reference
ON: Turkey L., Sault Ste. Marie area	1983 - 1985	Mink *Mustela vison*	NR	Kidney	14	1.86 ± 0.73 0.80 to 2.82	NR	NR	Wren et al. 1986
ON: Turkey L., Sault Ste. Marie area	1983 - 1985	Mink *Mustela vison*	NR	Muscle	14	1.04 ± 0.46 0.44 to 1.91	NR	NR	Wren et al. 1986
ON: Turkey L., Sault Ste. Marie area	1983 - 1985	Mink *Mustela vison*	NR	Brain	16	2.36 ± 1.63 1.12 to 7.50	NR	NR	Wren et al. 1986
ON: Turkey L., Sault Ste. Marie area	1983 - 1985	Otter *Lutra canadensis*	NR	Liver	8	1.99 ± 0.52 1.18 to 2.60	NR	NR	Wren et al. 1986
ON: Turkey L., Sault Ste. Marie area	1983 - 1985	Otter *Lutra canadensis*	NR	Kidney	6	1.90 ± 0.76 1.42 to 2.52	NR	NR	Wren et al. 1986
ON: Turkey L., Sault Ste. Marie area	1983 - 1985	Otter *Lutra canadensis*	NR	Muscle	7	1.02 ± 0.34 0.36 to 1.39	NR	NR	Wren et al. 1986
QC: natural areas of N. Québec	1990s	Mink *Mustela vison*	NR	Liver	38	4.85 1.17 to 15.60	NR	NR	Bélanger and Larivière 1997
QC: natural areas of N. Québec	1990s	Mink *Mustela vison*	NR	Kidney	38	2.45 0.73 to 6.32	NR	NR	Bélanger and Larivière 1997
QC: natural areas of N. Québec	1990s	Mink *Mustela vison*	NR	Brain	37	0.93 0.27 to 2.57	NR	NR	Bélanger and Larivière 1997
QC: Great Whale area, Northern. Québec	1989 - 1990	Mink *Mustela vison*	NR	Muscle	6	2.40 ± 2.24	NR	NR	Langlois et al. 1995

NR= Not Reported
ND = Not Detected

APPENDIX B-6: LEVELS OF TOTAL MERCURY AND METHYLMERCURY IN MARINE MAMMALS IN THE CANADIAN ENVIRONMENT.

Location	Year	Species	Life Stage, Age or Sex	Tissue	N	Total Hg μg·g^{-1} ww	MeHg μg·g^{-1} ww	% MeHg	Reference
Arctic: Eastern	1990	Beluga whale *Delphinapterus leucas*	NR	Liver	66	6	NR	NR	Wagemann et al. 1995
Arctic: Eastern	1993-1994	Beluga whale *Delphinapterus leucas*	2.5 to 24.5 years	Liver	73	10.2 ± 8.0 1.24 to 38.6	NR	NR	Wagemann 1995; Wagemann et al. 1996
Arctic: Eastern	1993-1994	Beluga whale *Delphinapterus leucas*	2.5 to 24.5 years	Muktuk	45	0.59 ± 0.22 0.32 to 1.37	NR	NR	Wagemann 1995; Wagemann et al. 1996
Arctic: Eastern	1993-1994	Beluga whale *Delphinapterus leucas*	2.5 to 24.5 yrs	Muscle	74	1.04 ± 0.43 0.44 to 2.7	NR	NR	Wagemann 1995; Wagemann et al. 1996
Arctic: Eastern	1983	Narwhal *Monodon monoceros*	NR	Liver	38	5.9	NR	NR	Wagemann et al. 1995
Arctic: Eastern	1992-1994	Narwhal *Monodon monoceros*	280-470 cm	Kidney	55	1.93 ± 1.12 0.29 to 6.92	NR	NR	Wagemann et al. 1996
Arctic: Eastern	1992-1994	Narwhal *Monodon monoceros*	280-470 cm	Liver	55	10.8 ± 8.05 0.32 to 37.2	NR	NR	Wagemann et al. 1996
Arctic: Eastern	1992-1994	Narwhal *Monodon monoceros*	280-470 cm	Muktuk	48	0.59 ± 0.18 0.16 to 1.27	NR	NR	Wagemann et al. 1996
Arctic: Eastern	1992-1994	Narwhal *Monodon monoceros*	280-470 cm	Muscle	56	1.03 ± 0.37 0.41 to 1.94	NR	NR	Wagemann et al. 1996
Arctic: Eastern	1989	Ringed seal *Phoca hispida*	NR	Liver	54	8.8	NR	15-30[a]	Wagemann et al. 1995

Location	Year	Species	Life Stage, Age or Sex	Tissue	N	Total Hg μg·g^{-1} ww	MeHg μg·g^{-1} ww	% MeHg	Reference
Arctic: Eastern	1989-1993	Ringed seal *Phoca hispida*	Young of the year to 22 years	Liver	115	8.34 ± 7.03 0.36 to 38.7	NR	NR	Wagemann 1995; Wagemann et al. 1996
Arctic: Eastern	1989-1993	Ringed seal *Phoca hispida*	Young of the year to 22 years	Muscle	61	0.39 ± 0.17	NR	NR	Wagemann 1995; Wagemann et al. 1996
Arctic: Hudson Bay	1994	Atlantic walrus *Odobenus r. rosmarus*	NR	Liver	143	1.2	NR	NR	Wagemann et al. 1995
Arctic: location NR	NR	Narwhal *Monodon monoceros*	NR	Muscle	2	1.4, 1.6 (approx)^b	1.4, 1.6 (approx)^b	96	Wagemann et al. 1997
Arctic: location NR	NR	Ringed seal *Phoca hispida*	NR	Muscle	12	0.6 ± 0.4 (approx)^b	0.6 ± 0.4 (approx)^b	100	Wagemann et al. 1997
Arctic: location NR	NR	Beluga whale *Delphinapterus leucas*	NR	Muscle	2	2.3 (approx)^b	2.3 (approx)^b	98	Wagemann et al. 1997
Arctic: Pond inlet	1978-1979	Narwhal *Monodon monoceros*	262-485 cm	Kidney	55	1.71 ± 1.01 0.31 to 4.67	NR	NR	Wagemann et al. 1996
Arctic: Pond inlet	1978-1979	Narwhal *Monodon monoceros*	262-485 cm	Liver	38	6.10 ± 3.10 0.57 to 13.1	NR	NR	Wagemann et al. 1996
Arctic: Pond inlet	1978-1979	Narwhal *Monodon monoceros*	262-485 cm	Muscle	58	0.85 ± 0.28 0.22 to 1.59	NR	NR	Wagemann et al. 1996
Arctic: Western	1993-1994	Beluga whale *Delphinapterus leucas*	7 to 35 years	Liver	77	27.1 ± 24.7 0.31 to 116	NR	NR	Wagemann 1995; Wagemann et al. 1996

Location	Year	Species	Life Stage, Age or Sex	Tissue	N	Total Hg µg·g^{-1} ww	MeHg µg·g^{-1} ww	% MeHg	Reference
Arctic: Western	1993-1994	Beluga whale *Delphinapterus leucas*	7 to 35 years	Muktuk	65	0.78 ± 0.41 0.19 to 1.93	NR	NR	Wagemann 1995; Wagemann et al. 1996
Arctic: Western	1993-1994	Beluga whale *Delphinapterus leucas*	7 to 35 years	Muscle	76	1.34 ± 0.67 0.41 to 3.44	NR	NR	Wagemann 1995; Wagemann et al. 1996
Arctic: Western	1987-1993	Ringed seal *Phoca hispida*	Young of the year to 38 years	Liver[c]	145	32.6 ± 35.2 0.23 to 219	NR	NR	Wagemann 1995; Wagemann et al. 1996
Arctic: Western	1987-1993	Ringed seal *Phoca hispida*	Young of the year to 38 years	Muscle	133	0.41 ± 0.29	NR	NR	Wagemann 1995; Wagemann et al. 1996
Atlantic coast	1988	Harp seal *Phoca groenlandica*	Adult females & pups	Liver	20	10	NR	NR	Wagemann et al. 1995
Atlantic coast	1988	Pilot whale *Globicephala malaena*	NR	Liver	39	23	NR	NR	Wagemann et al. 1995
Atlantic coast	1988	White-beaked dolphin *Lagenorhynchus albirostris*	NR	Liver	27	0.6	NR	NR	Wagemann et al. 1995
QC: Great Whale area, Northern Québec	1989-1990	Beluga whale *Delphinapterus leucas*	NR	Brain Liver Muscle	6	2.63 ± 2.87 20.34 ± 19.60 2.60 ± 2.06	NR 7.26 ± 5.21 1.01 ± 0.58	NR 36% 39%	Langlois et al. 1995
QC: Great Whale area, Northern Québec	1989-1990	Freshwater seal *Phoca vitulina*[d]	NR	Liver Muscle	2	28.8 ± 37.06 1.10 ± 0.37	4.55 ± 4.6 1.04 ± 0.37	16% 95%	Langlois et al. 1995

Location	Year	Species	Life Stage, Age or Sex	Tissue	N	Total Hg µg·g⁻¹ ww	MeHg µg·g⁻¹ ww	% MeHg	Reference
QC: Great Whale area, Northern Québec	1989-1990	Ringed seal *Phoca hispida*	NR	Brain Kidney Liver Muscle	8	0.19 ± 0.09 0.49 ± 0.07 5.12 ± 4.64 0.32 ± 0.19	NR NR 1.01 ± 0.87 0.11 ± 0.08	NR NR 20% 34%	Langlois et al. 1995
QC: St. Lawrence R.	1982-1987	Beluga whale *Delphinapterus leucas*	YOY to 30 years	Muscle	9	2.46 ± 1.46 0.89 to 5.54	NR	NR	Wagemann et al. 1996
QC: St. Lawrence R.	1982-1987	Beluga whale *Delphinapterus leucas*	YOY to 30 years	Liver	30	33.6 ± 43.0 0.38 to 202	NR	NR	Wagemann et al. 1996

NR = Not Reported

Notes:

[a] Or lower than 10% if calculated using regression rather than an average of percentages (Wagemann et al. 1997; R. Wagemann 1999, DFO, pers. com.).

[b] Numbers estimated from graphical presentation.

[c] Ringed seal liver, but not beluga liver, is consumed by Indigenous People. Most Hg in livers was inorganic Hg.

[d] The freshwater seal is a population of harbour seals captive of inland waters in Des Loups-Marins Lake and vicinity, 74° 00W, 56° 30N approx (L-G de Repentigny 1999, Environment Canada, pers.com.)

APPENDIX B-7:LEVELS OF TOTAL MERCURY AND METHYLMERCURY IN BIRDS IN THE CANADIAN ENVIRONMENT.

Location	Year	Species	Life Stage	Tissue	N	Total Hg $\mu g \cdot g^{-1}$ ww^{a}	MeHg $\mu g \cdot g^{-1}$ ww	% MeHg	Reference
BC: Lower mainland, Lower Fraser Valley	1990-1992	Bald eagle *Haliaeetus leucocephalus*	Eggs	Albumen and Yolk	6 7 11	0.186 to 0.358 0.174 to 0.296 NR	NR	NR NR 88	Elliott et al. 1996
BC: Vancouver Island, Crofton	1990-1992	Bald eagle *Haliaeetus leucocephalus*	Eggs	Albumen and Yolk	3	0.096 to 0.384	NR	NR	Elliott et al. 1996
BC: Vancouver Island, Johnstone Strait	1990-1992	Bald eagle *Haliaeetus leucocephalus*	Eggs	Albumen and Yolk	7	0.236 to 0.367	NR	NR	Elliott et al. 1996
BC: Vancouver Island, Nanaimo	1990-1992	Bald eagle *Haliaeetus leucocephalus*	Eggs	Albumen and Yolk	8	0.110 to 0.198	NR	NR	Elliott et al. 1996
NB: Quoddy region	1984	Bonaparte's gull *Larus philadephia*	Adult	Feathers	8 4 4	4.14 ± 2.22 all 4.82 ± 2.99 F 3.45 ±1.15 M	NR	NR	Braune and Gaskin 1987
NB: Quoddy region, Bay of Fundy	1978-1984	Arctic tern *Sterna paradisaea*	Adult	Brain	31	0.094	NR	NR	Braune 1987b
NB: Quoddy region, Bay of Fundy	1978-1984	Arctic tern *Sterna paradisaea*	Adult	Kidney Liver Muscle	36	0.453 0.470 0.089	NR	NR	Braune 1987b

Location	Year	Species	Life Stage	Tissue	N	Total Hg $\mu g \cdot g^{-1}$ ww^a	MeHg $\mu g \cdot g^{-1}$ ww	% MeHg	Reference
NB: Quoddy region, Bay of Fundy	1978-1984	Black guillemots *Cepphus grylle*	Adult	Brain Kidney Liver Muscle	4	0.123 0.491 0.513 0.113	NR	NR	Braune 1987b
NB: Quoddy region, Bay of Fundy	1978-1984	Black-legged kittiwake *Rissa tridactyla*	Adult	Brain Kidney Liver Muscle	14 18 19 20	0.038 0.242 0.372 0.037	NR	NR	Braune 1987b
NB: Quoddy region, Bay of Fundy	1978-1984	Bonaparte's gull *Larus philadephia*	Adult	Brain	137	0.101	NR	NR	Braune 1987b
NB: Quoddy region, Bay of Fundy	1978-1984	Bonaparte's gull *Larus philadephia*	Adult	Kidney Liver Muscle	145	0.418 0.450 0.075	NR	NR	Braune 1987b
NB: Quoddy region, Bay of Fundy	1978-1984	Common eider duck *Somateria mollissima*	Adult	Kidney Liver Muscle	11	0.358 0.987 0.153	NR	NR	Braune 1987b
NB: Quoddy region, Bay of Fundy	1978-1984	Common tern *Sterna hirundo*	Adult	Brain	29	0.190	NR	NR	Braune 1987b
NB: Quoddy region, Bay of Fundy	1978-1984	Common tern *Sterna hirundo*	Adult	Kidney Liver Muscle	30	1.505 1.249 0.166	NR	NR	Braune 1987b

Location	Year	Species	Life Stage	Tissue	N	Total Hg $\mu g \cdot g^{-1}$ ww^a	MeHg $\mu g \cdot g^{-1}$ ww	% MeHg	Reference
NB: Quoddy region, Bay of Fundy	1978-1984	Double-crested cormorant *Phalacrocorax auritus*	Adult	Brain	1	0.360	NR	NR	Braune 1987b
NB: Quoddy region, Bay of Fundy	1978-1984	Double-crested cormorant *Phalacrocorax auritus*	Adult	Kidney Liver Muscle	3	5.35 7.05 0.606	NR	NR	Braune 1987b
NB: Quoddy region, Bay of Fundy	1978-1984	Herring gull *Larus argentatus*	Adult	Brain Kidney Liver Muscle	4	0.056 0.350 0.482 0.101	NR	NR	Braune 1987b
NB: Quoddy region, Bay of Fundy	1978-1984	Red-necked phalarope *Phalaropus lobatus*	Adult	Liver Muscle	12 13	0.225 0.046	NR	NR	Braune 1987b
NB: Quoddy region, Bay of Fundy	1978-1984	Six bird species	Adult	Feathers	968	0 to ~12 $\mu g \cdot g^{-1}$ dw	NR	NR	Braune 1987b
ON: Akwesasne Reserve, North-east of Cornwall	1991	Tree swallow *Tachycineta bicolor*	Eggs	Albumen and yolk	5	0.043	NR	NR	Bishop et al. 1995
ON: Algonquin/ Parry Sound	1970-1978	Common Loon *Gavia immer*	Eggs	Albumen and Yolk	61	0.81 to 1.11 avg	0.79	71	Frank et al. 1983

Location	Year	Species	Life Stage	Tissue	N	Total Hg $\mu g \cdot g^{-1}$ ww^a	MeHg $\mu g \cdot g^{-1}$ ww	% MeHg	Reference
ON: Algonquin/ Parry Sound	1971-1974	Common Loon *Gavia immer*	Healthy Adults & Juveniles	Brain Liver Muscle	128	0.44 to 0.57 1.92 to 6.35 0.79 to 1.14 (means)	NR	NR	Frank et al. 1983
ON: Algonquin/ Parry Sound	1971-1974	Common Loon *Gavia immer*	Emaciated Adults & Juveniles	Brain Liver Muscle	29	1.03 to 1.82 16.3 to 26.4 2.71 to 5.41 (means)	NR	NR	Frank et al. 1983
ON: Central Ontario	1992	Common Loon *Gavia immer*	Chick	Feather	17	2.31 ± 0.65	NR	NR	Scheuhammer et al. 1998
ON: Central Ontario	1992	Common Loon *Gavia immer*	Chick	Blood	35	0.14 ± 0.12	NR	NR	Scheuhammer et al. 1998
ON: Central Ontario	1992	Common Loon *Gavia immer*	Adult	Blood	22	2.06 ± 0.94	NR	NR	Scheuhammer et al. 1998
ON: Central Ontario	1992	Common Loon *Gavia immer*	Adult	Feather	20	13.3 ± 4.19	NR	NR	Scheuhammer et al. 1998
ON: Clay L. North-western Ontario	1971	American wigeon *Anas americana*	3 Immature 2 Adult	Breast	5	0.48 ± 0.30 0.3 to 0.9	NR	NR	Vermeer et al. 1973
ON: Clay L. North-western Ontario	1971	Blue-winged teal *Anas discors*	Immature	Breast	1	10.4	7.4	71	Vermeer et al. 1973

Location	Year	Species	Life Stage	Tissue	N	Total Hg $\mu g \cdot g^{-1}$ ww^a	MeHg $\mu g \cdot g^{-1}$ ww	% MeHg	Reference
ON: Clay L. North-western Ontario	1971	Common goldeneye *Bucephala clangula*	Immature	Breast	1	16.8	12.3	73	Vermeer et al. 1973
ON: Clay L. North-western Ontario	1971	Common merganser *Mergus merganser*	Immature	Breast	1	19.4	14.9	77	Vermeer et al. 1973
ON: Clay L. North-western Ontario	1971	Herring gull *Larus argentatus*	Egg	Albumen and yolk	14	8.4 ± 3.8 S.D	NR	NR	Vermeer et al. 1973
ON: Clay L. North-western Ontario	1971	Herring gull *Larus argentatus*	Egg	Albumen	4	12.0 ±7.8 S.D.	NR	NR	Vermeer et al. 1973
ON: Clay L. North-western Ontario	1971	Herring gull *Larus argentatus*	Egg	Yolk	4	2.3 ± 1.1 S.D.	NR	NR	Vermeer et al. 1973
ON: Clay L. North-western Ontario	1971	Hooded merganser *Lophodytes cucullatus*	Immature	Breast	1	17.9	17.8	99	Vermeer et al. 1973
ON: Clay L. North-western Ontario	1971	Mallard *Anas platyrhynchos*	Immature	Breast	1	0.16	0.11	69	Vermeer et al. 1973
ON: Detroit R.	1973-1992	Herring gull *Larus argentatus*	Eggs	Albumen and Yolk	Var	0.14 to 0.24	NR	NR	Koster et al. 1996
ON: Georgian Bay Wye Marsh	1991	Tree swallow *Tachycineta bicolor*	Eggs	Albumen and yolk	10	0.066	NR	NR	Bishop et al. 1995

Location	Year	Species	Life Stage	Tissue	N	Total Hg $\mu g \cdot g^{-1}$ ww[a]	MeHg $\mu g \cdot g^{-1}$ ww	% MeHg	Reference
ON: L. Erie 2 locations	1973-1992	Herring gull *Larus argentatus*	Eggs	Albumen and Yolk	Var	0.13 to 0.54	NR	NR	Koster et al. 1996
ON: L. Erie Mud Creek	1991	Tree swallow *Tachycineta bicolor*	Eggs	Albumen and yolk	10	0.079	NR	NR	Bishop et al. 1995
ON: L. Huron	1972-1973	Double-crested cormorant *Phalacrocorax auritus*	Eggs	Albumen and Yolk	UK[b]	0.32 to 0.83	NR	NR	Weseloh et al. 1983
ON: L. Huron 2 locations	1973-1992	Herring gull *Larus argentatus*	Eggs	Albumen and Yolk	Var	0.13 to 0.39	NR	NR	Koster et al. 1996
ON: L. Ontario	1971	Common tern *Sterna hirundo*	Eggs	Albumen and Yolk	UK[b]	1.08	NR	NR	Gilbertson 1974
ON: L. Ontario 6 locations	1973-1992	Herring gull *Larus argentatus*	Eggs	Albumen and Yolk	Var	0.18 to 0.88	NR	NR	Koster et al. 1996
ON: L. Ontario Long Point Tip	1991	Tree swallow *Tachycineta bicolor*	Eggs	Albumen and yolk	10	0.076	NR	NR	Bishop et al. 1995
ON: L. St. Clair	1973	Black-crowned night-heron, *Nycticorax nycticorax*	Eggs	Albumen and Yolk	UK[b]	0.31 to 0.73	NR	NR	Stendell et al. 1976
ON: L. St. Clair	1973	Common tern *Sterna hirundo*	Eggs	Albumen and Yolk	UK[b]	0.73 to 1.09	NR	NR	Stendell et al. 1976

Location	Year	Species	Life Stage	Tissue	N	Total Hg µg·g^{-1} ww^{a}	MeHg µg·g^{-1} ww	% MeHg	Reference
ON: L. St. Clair	1973	Great egret *Casmerodius albus*	Eggs	Albumen and Yolk	UK^{b}	0.24 to 0.43	NR	NR	Stendell et al. 1976
ON: L. St. Clair	1973	Mallard *Anas platyrhynchos*	Eggs	Albumen and Yolk	UK^{b}	0.05 to 0.20	NR	NR	Stendell et al. 1976
ON: L. Superior 4 locations	1973-1992	Herring gull *Larus argentatus*	Eggs	Albumen and Yolk	Var	0.15 to 0.59	NR	NR	Koster et al. 1996
ON: L. Superior	1973	Herring gull *Larus argentatus*	Eggs	Albumen and Yolk	UK^{b}	0.41 to 0.62	NR	NR	Ryder 1974
ON: L. Superior	1973	Ring-billed gull *Larus delawarensis*	Eggs	Albumen and Yolk	UK^{b}	0.45 to 0.75	NR	NR	Ryder 1974
ON: Lakes (12) disconnected from Wabigoon-English R.	1974-1975	Common Loon *Gavia immer*	Eggs	Albumen and Yolk	13	0.59 ± 0.10	0.58 ± 0.10	99	Barr 1985
ON: Lakes (12) disconnected from Wabigoon-English R.	1976	Common Loon *Gavia immer*	Adult	Brain Liver Muscle	10	0.4 ± 0.1 8.8 ± 4.6 1.2 ± 0.6	0.2 ± 0.4	4	Barr 1985
ON: Lakes (12) disconnected from Wabigoon-English R.	1976	Common Loon *Gavia immer*	Chick	Brain Liver Muscle	4	0.4 ± 0.1 0.8 ± 0.3 0.4 ± 0.1	0.7 ± 0.2	87	Barr 1985

Location	Year	Species	Life Stage	Tissue	N	Total Hg µg·g^{-1} ww^{a}	MeHg µg·g^{-1} ww	% MeHg	Reference
ON: Lakes (6) downstream of Wabigoon R, Dryden	1974-1975	Common Loon *Gavia immer*	Eggs	Albumen and Yolk	5	1.39 ± 0.55	1.34 ± 0.53	97	Barr 1985
ON: Lakes (6) downstream of Wabigoon R, Dryden	1976	Common Loon *Gavia immer*	Adult	Brain Liver Muscle	5	1.5 ± 0.6 29.7 ± 12.4 4.6 ± 1.6	1.5 ± 2.1	9	Barr 1985
ON: Lakes (10) upstream of Maynard Falls on English R.	1974-1975	Common Loon *Gavia immer*	Eggs	Albumen and Yolk	10	0.54 ± 0.09	0.53 ± 0.10	97	Barr 1985
ON: Lakes (10) upstream of Maynard Falls on English R.	1976	Common Loon *Gavia immer*	Adult	Brain Liver Muscle	12	0.4 ± 0.1 5.1 ± 3.2 1.2 ± 0.5	1.0 ± 0.7	27	Barr 1985
ON: Lakes (10) upstream of Maynard Falls on English R.	1976	Common Loon *Gavia immer*	Chick	Brain Liver Muscle	6	0.3 ± 0.1 0.9 ± 0.4 0.4 ± 0.2	0.8 ± 0.4	92	Barr 1985
ON: Lakes (6) Upstream of Wabigoon-English R.	1974-1975	Common Loon *Gavia immer*	Eggs	Albumen and Yolk	6	0.72 ± 0.23	0.71 ± 0.22	99	Barr 1985
ON: Lakes (6) Upstream of Wabigoon-English R.	1976	Common Loon *Gavia immer*	Adult	Brain Liver Muscle	4	1.2 ± 0.6 16.7 ± 10.9 3.4 ± 2.0	1.1 ± 2.2	7	Barr 1985
ON: Niagara R. Un-named island	1973-1992	Herring gull *Larus argentatus*	Eggs	Albumen and Yolk	Var	0.15 to 0.35	NR	NR	Koster et al. 1996

Location	Year	Species	Life Stage	Tissue	N	Total Hg µg·g^{-1} ww^{a}	MeHg µg·g^{-1} ww	% MeHg	Reference
QC: Great Whale area, Northern Québec	1989-1990	Common merganser *Mergus merganser*	Adult	Liver Muscle	13	17.53 ± 12.06 1.27 ± 0.48	NR	NR	Langlois et al. 1995
QC: Great Whale area, Northern Québec	1989-1990	Herring gull *Larus argentatus*	Adult	Muscle	28	1.03 ± 0.80	NR	NR	Langlois et al. 1995
QC: Great Whale area, Northern Québec	1989-1990	Herring gull *Larus argentatus*	Adult	Liver	27	2.91 ± 2.35	NR	NR	Langlois et al. 1995
QC: Great Whale area, Northern Québec	1989-1990	Herring gull *Larus argentatus*	Eggs	NR	2	0.28 ± 0.12	NR	NR	Langlois et al. 1995
QC: Lakes & rivers North-west Québec	1989-1991	Osprey *Pandion haliaetus*	Eggs	Whole	33	0.2 ± 0.1	NR	NR	DesGranges et al. 1998
QC: Lakes & rivers North-west Québec	1989-1991	Osprey *Pandion haliaetus*	Nestling	Feather	63	7.0 ± 4.3	NR	NR	DesGranges et al. 1998
QC: Lakes & rivers North-west Québec	1989-1991	Osprey *Pandion haliaetus*	Adult	Feather	29	16.5 ± 12.8	NR	NR	DesGranges et al. 1998

Location	Year	Species	Life Stage	Tissue	N	Total Hg µg·g^{-1} ww^{a}	MeHg µg·g^{-1} ww	% MeHg	Reference
QC: Lakes & rivers North-west Québec	1989-1991	Osprey *Pandion haliaetus*	NR	Stomach Content	6	0.3 ± 0.2	NR	NR	DesGranges et al. 1998
QC: Nottaway-Broadback-Rupert area, South-east coast James Bay	1990-1991	Common merganser *Mergus merganser*	Adult	Muscle	19	1.41 ± 1.26	NR	NR	Langlois et al. 1995
QC: Nottaway-Broadback-Rupert area, South-east coast James Bay	1990-1991	Common merganser *Mergus merganser*	Adult	Liver	15	10.9 ± 7.5	NR	NR	Langlois et al. 1995
QC: Nottaway-Broadback-Rupert area, South-east coast James Bay	1990-1991	Herring gull *Larus argentatus*	Adult	Muscle	13	1.59 ± 1.32	NR	NR	Langlois et al. 1995
QC: Reservoirs North-west Québec	1989-1991	Osprey *Pandion haliaetus*	Eggs	Whole	18	0.2 ± 0.1	NR	NR	DesGranges et al. 1998
QC: Reservoirs North-west Québec	1989-1991	Osprey *Pandion haliaetus*	Nestling	Feather	78	37.4 ± 20.1	NR	NR	DesGranges et al. 1998
QC: Reservoirs North-west Québec	1989-1991	Osprey *Pandion haliaetus*	Adult	Feather	31	58.1 ± 51.3	NR	NR	DesGranges et al. 1998

Location	Year	Species	Life Stage	Tissue	N	Total Hg $\mu g \cdot g^{-1}$ ww[a]	MeHg $\mu g \cdot g^{-1}$ ww	% MeHg	Reference
QC: Reservoirs North-west Québec	1989-1991	Osprey *Pandion haliaetus*	NR	Stomach Content	6	0.8 ± 0.33	NR	NR	DesGranges et al. 1998

NR = Not Reported

Var = Variation in the number of samples

Notes:

[a] Unless noted otherwise, i.e., dw = dry weight.

[b] UK = unknown, information cited from Koster et al. 1996

APPENDIX B-8: LEVELS OF TOTAL MERCURY AND METHYLMERCURY IN SELECTED NON-BIOLOGICAL MEDIA IN THE CANADIAN ENVIRONMENT.

Location	Year	Media	N	Total Hg ng·L^{-1} or µg·g^{-1} dw^{a}	MeHg ng·L^{-1} or µg·g^{-1} dw	% MeHg	Reference
ON/QC: Black L.	1987	Water, lake	5	3.5	1.3	37%	Schintu et al. 1989
ON/QC: Gatineau R.	1987	Water, river	5	11.4	4.1	36%	Schintu et al. 1989
ON/QC: Ottawa R.	1987	Water, river	5	9.0	2.3	26%	Schintu et al. 1989
ON: Experimental Lakes Area	1992	Rain, August & Sept. samples	4	4.62	0.017	0.4%	St. Louis et al. 1995
ON: Experimental Lakes Area	1993	Rain, May – August	6	5.33	0.049	0.9%	St. Louis et al. 1995
ON: Experimental Lakes Area	1995	Peat porewater	NR	6	0.6	10%	Moore et al. 1995
ON: Experimental Lakes Area	1993	Snow on lake surface	2	0.95	0.008	0.8%	St. Louis et al. 1995
ON: Experimental Lakes Area	1994	Snow on lake surface	2	1.96	0.032	1.7%	St. Louis et al. 1995
ON: Experimental Lakes Area	1992	Snow, Oct. & Nov. samples	2	3.58	0.104	2.9%	St. Louis et al. 1995
ON: Experimental Lakes Area	1991	Water from upland area	9	13.11 ± 0.67	0.03 ± 0.006	0.2%	St. Louis et al. 1994
ON: Experimental Lakes Area	1991	Water from wetland dominated area	19	5.02 ± 0.36	0.626 ± 0.126	13.3%	St. Louis et al. 1994
ON: Experimental Lakes Area	1991	Water from wetland/upland area#1	15	11.77 ± 0.73	0.176 ± 0.012	1.6%	St. Louis et al. 1994
ON: Experimental Lakes Area	1991	Water from wetland/upland area#2	9	11.40 ± 0.87	0.228 ± 0.033	2.0%	St. Louis et al. 1994
ON: Experimental Lakes Area	1991-1993	Water, Pond 979 before flooding	NR	2.6 ± 1.5	0.09 ± 0.04	4.4%	Kelly et al. 1995

Location	Year	Media	N	Total Hg ng·L^{-1} or µg·g^{-1} dw^{a}	MeHg ng·L^{-1} or µg·g^{-1} dw	% MeHg	Reference
ON: Experimental Lakes Area	1991-1993	Water, Pond 979 after flooding	NR	4.5 ± 1.7	1.38 ± 0.51	31%	Kelly et al. 1995
ON: Experimental Lakes Area	1991	Water, upland/wetland & lake area	16	1.69 ± 0.27	0.040 ± 0.003	4.4%	St. Louis et al. 1994
ON: Experimental Lakes Area	1991	Wet deposition	5	3.81 ± 0.85	0.019 ± 0.004	0.6%	St. Louis et al. 1994
ON: St. Lawrence R., Cornwall area	1994	Surface sediments from riverine wetlands	69	0.015 to 0.882	NR	1%	Thompson 1996
ON: Wabigoon/English R.	1978-1979^{b}	Water, Clay L.	26	NR	1.3	NR	Parks et al. 1989
ON: Wabigoon/English R.	1978-1979^{b}	Water, English R., Ball L. outflow	12	NR	0.22	NR	Parks et al. 1989
ON: Wabigoon/English R.	1978-1979^{b}	Water, Wabigoon L.	25	NR	0.27	NR	Parks et al. 1989
ON: Wabigoon/English R.	1978-1979^{b}	Water, Wabigoon R., Clay L. outflow	50	NR	1.3	NR	Parks et al. 1989
ON: Wabigoon/English R.	1978-1979^{b}	Water, Wabigoon R., inflow to Wainwright Dam	25	NR	0.43	NR	Parks et al. 1969
ON: Wetlands (22) South-central Ontario	1993	Surface sediments from various types of wetlands	198	0.081 to 0.316	NR	1%	Thompson 1996
QC: Great Whale area, Northern Québec	1989-1990	Sediments	12	0.07 ± 0.05	NR	NR	Langlois et al. 1995
QC: Great Whale area, Northern Québec	1989-1990	Seston – fall samples	7	0.11 ± 0.07	0.02 ± 0.02	18.2%	Langlois et al. 1995
QC: Great Whale area, Northern Québec	1989-1990	Water – summer samples	22	4.8 ± 6.62	0.453 ± 0.37	9.4%	Langlois et al. 1995
QC: Natural lakes, Northern Québec	1994-1996	Water – dissolved Hg	NR	1.51 ± 0.06 0.4 - 2.6	0.049 ± 0.004 0.018 - 0.115	3.2%	Lucotte et al. 1998

Location	Year	Media	N	Total Hg ng·L^{-1} or µg·g^{-1} dw[a]	MeHg ng·L^{-1} or µg·g^{-1} dw	% MeHg	Reference
QC: R. Bourassa and Laforge-1 Reservoirs	1994-1996	Water	87	2.35 ± 0.12 0.96 - 5.34	0.28 ± 0.02 0.03-0.85	12%	Thérien and Morrison 1998
QC: Lakes near R. Bourassa and Laforge-1 Reservoirs	1994-1996	Water	30	1.51 ± 0.06 0.4 - 2.6	0.05 ± 0.004 0.02 - 0.11	3%	Thérien and Morrison 1998
QC: La Grande 2 Reservoirs	1992	Soils, flooded, 0-4 cm depth	NR	0.129	0.0085	6.6%	Mucci et al. 1995
QC: La Grande 2 Reservoirs	1992	Soils, flooded, 11-19 cm depth	NR	0.032	0.0019	5.9%	Mucci et al. 1995
QC: La Grande 2 Reservoirs	1992	Soils, flooded, 4-11 cm depth	NR	0.142	0.043	30%	Mucci et al. 1995
QC: Northern reservoirs/lake	1992	Sediments, surface	NR	0.036 to 0.059	0.0001 to 0.0006	1%	Tremblay et al. 1996
QC: Nottaway-Broadback-Rupert area, SE coast James Bay	1990-1991	Water – summer samples	25	3.07 ± 1.71	0.94 ± 0.54	31%	Langlois et al. 1995
QC: Nottaway-Broadback-Rupert area, SE coast James Bay	1990-1991	Water – winter samples	20	4.45 ± 1.73	0.46 ± 0.83	10.3%	Langlois et al. 1995
SK: Pasqua L.	1982[b]	Water	4	51.6	0.081	0.16%	Jackson 1993

NR = Not Reported

Notes:

[a] ng·L^{-1} is unit for water-based media; µg·g^{-1} is unit for solid media.

[b] Samples taken at a time prior to realization that "ultra-clean" techniques were necessary.

APPENDIX C-1: SUMMARY OF DATA ON THE ACUTE OR CHRONIC TOXICITY OF ORALLY-ADMINISTERED METHYLMERCURY IN MAMMALS.

Species	Life Stage	Sex	N	Daily dose mg·kg^{-1} in diet µg·kg^{-1} bw·d^{-1}	Length of exposure	Total dose mg·kg^{-1} bw	[Tissue] mg·kg^{-1} ww	Endpoint and Effects	Reference
Cat *Felis catus*	Adult	NR	NR	0.25 (fed 5 of 7 d) 250	90 d	22.5 (16.1/cat)	NR	First convulsions at 68d; mean survival 78d	Eaton et al. 1980
Cat *Felis catus*	Adult	NR	NR	0.55 ± 0.29 S.E.	188 d	18-29/cat	15.1 Muscle 170 Hair	Behavioural effects following tuna fish diet	Houpt et al. 1988
Cat *Felis catus*	Adult	NR	NR	0.5	7-11 mos	NR	NR	Proliferation of smooth ER; degeneration of hepatic mitochondria (tuna fish diet)	Chang et al. 1974
Cat *Felis catus*	Adult	NR	NR	0.05, 0.14, 0.3, 0.76, 1.2, 2.9 3, 8.4, 20, 46, 74, 176	39 weeks (273 d)	NR	NR	Neurologic damage at 46 µg·kg^{-1} bw (0.76 mg·kg^{-1}) in diet of fish	Charbonneau et al. 1976
Crab-eating macaque *Macaca fascicularis*	0 - 7 years	NR	5	NR 50	7 years	128	NR	Lost sensitivity to touch and tactile response	Rice 1989
Crab-eating macaque *Macaca fascicularis*	0 - 13 years	NR	5	NR 50	Exposed age 0 to 7; Not exposed age 7 to 13	128	NR	Clumsier and slower to react than controls	Rice 1989

Species	Life Stage	Sex	N	Daily dose mg·kg^{-1} in diet µg·kg^{-1} bw·d^{-1}	Length of exposure	Total dose mg·kg^{-1} bw	[Tissue] mg·kg^{-1} ww	Endpoint and Effects	Reference
Crab-eating macaque *Macaca fascicularis*	Adult	F	7	NR 90	1 year	32.4	NR	4/7 suffered neurologic damage	Burbacher et al. 1984
Crab-eating macaque *Macaca fascicularis*	Adult	NR	NR	NR 0.4 – 50	150 d	0.06 to 7.5	NR	No clinical symptoms, no difference in cholinesterase activity	Petruccioli and Turillazzi 1991
Harp seal *Phoca groenlandica*	Adult	NR	NR	0.25 NR	60 d	NR	8.9 Blood	Decline in appetite and body weight	Ronald et al. 1977
Harp seal *Phoca groenlandica*	Adult	NR	NR	0.25 NR	90 d	NR	12.5 Blood	Reduced activity after 60 d	Ronald et al. 1977
Harp seal *Phoca groenlandica*	Adult	NR	NR	25	26 d	NR	21.3 Blood	Lethargy; weight loss; death on day 20 to 26	Ronald et al. 1977
Mice *Mus musculus* Strain: B6C3-F1	Adult	M/F	120	0.4 40	2 years	28.8	NR	NOAEL	Mitsumori et al. 1990
Mice *Mus musculus* Strain: B6C3-F1	Adult	M/F	120	2 170	2 years	122	NR	LOAEL - chronic kidney disease	Mitsumori et al. 1990
Mice *Mus musculus* Strain: B6C3-F1	Adult	F	60	10 830	2 years	598	NR	Posterior paralysis in 3/60 after 80 weeks, brain necrosis; chronic kidney disease	Mitsumori et al. 1990

Species	Life Stage	Sex	N	Daily dose mg·kg^{-1} in diet µg·kg^{-1} bw·d^{-1}	Length of exposure	Total dose mg·kg^{-1} bw	[Tissue] mg·kg^{-1} ww	Endpoint and Effects	Reference
Mice *Mus musculus* Strain: B6C3-F1	Adult	M	60	10 830	2 years	598	NR	Posterior paralysis in 33/60 after 59 weeks, decreased bw gain; brain necrosis; chronic kidney disease	Mitsumori et al. 1990
Mink *Mustela vison*	Adult	12F 4M	16	1.0 100 M; 180 F	11.5 wks (81 d)	8.1 M 14.6 F	15.3 F Brain	4/12 F died, dose cut in half for remainder (see next table row). Brain lesions in F that died.	Wren et al. 1987a
Mink *Mustela vison*	Adult	4F 3M	7	1.0 reduced to 0.5[a] 100 to 50 M; 180 to 90 F	11.5 wks @ 1.0 15 wks @ 0.5	8.1 +5.3 = 13.4 M 14.6 + 9.5 =24.1 F	18 M Brain 4.2 F Brain	No clinical effects (C. Wren, pers. com.) - elevated Hg in offspring livers measured.	Wren et al. 1987a
Mink *Mustela vison*	Adult	NR	NR	5.0 500 M; 700 F[b]	30-37 d	15-18.5 M 21-26 F	25.2 Muscle 20 Brain	LT_{50} - Ataxia, anorexia, paralysis, death	Aulerich et al. 1974
Mink *Mustela vison*	Adult	F	10	0.25 38[c]	120 d	4.5	0.5 Brain	No clinical or pathologic effect	Wobeser et al. 1976a
Mink *Mustela vison*	Adult	F	10	0.33 53	120 d	6.4	3.4 Brain	No clinical or pathologic effect (NOAEL)	Wobeser et al. 1976a
Mink *Mustela vison*	Adult	F	5	<0.1 <16	93 d	<1.5	0.2 Muscle 0.1 Brain	Control	Wobeser et al. 1976b
Mink *Mustela vison*	Adult	F	5	1.1 176	93 d	16.4	7.8 Muscle 8.2 Brain	LOAEL: pale livers, nerve tissue lesions, 2/5 showed reduced movement.	Wobeser et al. 1976b

Species	Life Stage	Sex	N	Daily dose mg·kg^{-1} in diet µg·kg^{-1} bw·d^{-1}	Length of exposure	Total dose mg·kg^{-1} bw	[Tissue] mg·kg^{-1} ww	Endpoint and Effects	Reference
Mink *Mustela vison*	Adult	F	5	1.8 288	79 d	22.8	4.9 Muscle 18.1 Brain	Anorexia, ataxia at 50-80 d; death at 59-79 d	Wobeser et al. 1976b
Mink *Mustela vison*	Adult	F	5	4.8 768	36 d	27.6	14.1 Muscle 10.5 Brain	Anorexia, ataxia at 23-32 d; death at 26-36 d	Wobeser et al. 1976b
Mink *Mustela vison*	Adult	F	5	8.3 1328	26 d	34.5	17.4 Muscle 13.3 Brain	Anorexia, ataxia at 16-21 d; death at 19-26 d	Wobeser et al. 1976b
Mink *Mustela vison*	Adult	F	5	15.0 2250	20 d	45.0	22 Muscle 15.6 Brain	Anorexia, ataxia at 16-18 d; death at 18-20 d	Wobeser et al. 1976b
Mink *Mustela vison*	6 mos.	F	20	0.1 16^{c}	101 d	16.2	NR	No effect	Chamberland et al. 1996
Mink *Mustela vison*	6 mos.	F	20	0.5 80	101 d	8.1	NR	No effect reported.	Chamberland et al. 1996
Mink *Mustela vison*	6 mos.	F	20	0.9 144	101 d	14.5	NR	Lethal to >50% between 80-101 days; signs of neurotoxicity; minor renal damage.	Chamberland et al. 1996
Otter *Lutra canadensis*	Adult	M	3	2.0 90	159-213 d	14.6 to 21.4	12 Muscle 13.3 Brain	Lethal to 2/3; Anorexia in 1/3 and ataxia in 2/3 otters (survivors euthanized)	O'Connor and Nielsen 1981
Otter *Lutra canadensis*	Adult	M	3	4.0 170	113-122 d	17.9 to 21.3	16.3 Muscle 18.5 Brain	Anorexia and ataxia in 3 of 3 otters (euthanized); nephrotic and neurologic lesions	O'Connor and Nielsen 1981

Species	**Life Stage**	**Sex**	**N**	**Daily dose mg·kg^{-1} in diet µg·kg^{-1} bw·d^{-1}**	**Length of exposure**	**Total dose mg·kg^{-1} bw**	**[Tissue] mg·kg^{-1} ww**	**Endpoint and Effects**	**Reference**
Otter *Lutra canadensis*	Adult	M	3	8.0 370	45 - 57 d	16.1 to 22.4	15.2 Muscle 14 Brain	Lethal to 1/3 (survivors euthanized); nephrotic and neurologic lesions	O'Connor and Nielsen 1981
Otter *Lutra canadensis*	Adult	M	2	0 0	192-229 d	0	0.77 Muscle 0.38 Brain	Control	O'Connor and Nielsen. 1981
Rat *Rattus* spp. Strain: unknown	Adult	F	25	2.5 100	2 years	73	NR	56% survival at end, 6% lower weight gain (60 weeks); 76% survival controls	Verschuuren et al. 1976
Rat *Rattus* spp. Strain: unknown	Adult	M	25	2.5 100	2 years	73	NR	48% survival at end; 72% survival in controls	Verschuuren et al. 1976
Rat *Rattus norvegicus* Strain: Sprague-Dawley	Adult	F	56	NR 340	104 wks	248	NR	30% survival at this dosage	Mitsumori et al. 1983, 1984
Rat *Rattus norvegicus* Strain: Sprague-Dawley	Adult	M	56	NR 280	104 wks	204	NR	10% survival at this dosage	Mitsumori et al. 1983, 1984

Species	Life Stage	Sex	N	Daily dose $mg{\cdot}kg^{-1}$ in diet $\mu g{\cdot}kg^{-1}$ $bw{\cdot}d^{-1}$	Length of exposure	Total dose $mg{\cdot}kg^{-1}$ bw	[Tissue] $mg{\cdot}kg^{-1}$ ww	Endpoint and Effects	Reference
Rat *Rattus norvegicus* Strain: Wistar rat	Adult	F	50	NR 50	26 mos	3.9	NR	NOAEL - females	Munro et al. 1980
Rat *Rattus norvegicus* Strain: Wistar rat	Adult	M	50	NR 50	26 mos	3.9	NR	NOAEL - males	Munro et al. 1980
Rat *Rattus norvegicus* Strain: Wistar rat	Adult	F	50	NR 250	26 mos	19.5	NR	Reduced bw gains; nerve damage	Munro et al. 1980
Rat *Rattus norvegicus* Strain: Wistar rat	Adult	M	50	NR 250	26 mos	19.5	NR	Overt neurotoxicity; decreased hemoglobin and hematocrit, reduced bw gains, mortality; nerve damage; severe kidney damage.	Munro et al. 1980
Rat *Rattus norvegicus* Strain: Wistar rat	Adult	F	4	NR 5 - 50	4 d	NR	NR	Fed days 6-9 of gestation; 4 mo old offspring showed operant behaviour deficits - LOAEL 10 $\mu g{\cdot}kg^{-1}$ bw/d	Bornhausen et al. 1980
Rhesus monkey *Macaca mulatta*	Adult	NR	NR	NR 125	3.5 mos	13.1	NR	Liver and kidney histological changes	Rice et al. 1989

Species	Life Stage	Sex	N	Daily dose $mg \cdot kg^{-1}$ in diet $\mu g \cdot kg^{-1}$ $bw \cdot d^{-1}$	Length of exposure	Total dose $mg \cdot kg^{-1}$ bw	[Tissue] $mg \cdot kg^{-1}$ ww	Endpoint and Effects	Reference
Rhesus monkey *Macaca mulatta*	Adult	NR	NR	NR 80	7-12 mos	17 to 29	NR	Liver and kidney histological changes	Rice et al. 1989
Rhesus monkey *Macaca mulatta*	Adult	NR	NR	NR 100	10 mos	30	NR	Liver normal; kidney effects evident	Rice et al. 1989
Rhesus monkey *Macaca mulatta*	Adult	NR	NR	NR 80-100	15 mos	36 to 45	NR	Liver normal; kidney effects evident	Rice et al. 1989
Rhesus monkey *Macaca mulatta*	Adult	NR	NR	NR 90	10 mos	27	NR	Liver normal; kidney effects evident	Rice et al. 1989

NR = Not Reported

Notes:

[a] 1 $mg \cdot kg^{-1}$ fed daily for 11.5 weeks, then fed every other day for remainder of 6 month study (~15 weeks) (1.0 $mg \cdot kg^{-1}$ daily dose caused mortality to 3 F; 6 animals in discomfort were euthanized).

[b] Calculated using food consumption rates of 0.1 and 0.14 $kg \cdot kg^{-1}$ bw per day for males (M) and females (F), respectively (Aulerich et al. 1974)

[c] Calculated using a food consumption rate of 0.16 $kg \cdot kg^{-1}$ bw per day for female captive mink (Bleavins and Aulerich 1981)

APPENDIX C-2: SUMMARY OF DATA ON THE REPRODUCTIVE AND DEVELOPMENTAL TOXICITY OF ORALLY-ADMINISTERED METHYLMERCURY IN MAMMALS.

Species	**Life Stage**	**Sex**	**N**	**Daily dose μg·kg^{-1} bw**	**Duration of exposure**	**Total dose mg·kg^{-1} bw**	**Endpoint and Effects**	**Reference**
Crab-eating macaque *Macaca fascicularis*	Adult	F	11	50 - 70	Reproductive cycle	NR	Mothers dosed through pregnancy; Visual recognition deficits in 35d old infants	Gunderson et al. 1986
Crab-eating macaque *Macaca fascicularis*	Adult	F	7	50	Reproductive cycle	NR	2/7 aborted - not statistically significant	Burbacher et al. 1984
Crab-eating macaque *Macaca fascicularis*	Adult	F	7	90	Reproductive cycle		2/7 aborted, 3/7 no conception, not statistically significant	Burbacher et al. 1984
Mink *Mustela vison*	Adult	4F 3M	7	1.0 reduced to 0.5[a] 100 to 50 M; 180 to 90 F	11.5 wks @ 1.0 15 wks @ 0.5	8.1 +5.3 = 13.4 M 14.6 + 9.5 =24.1 F	Fertility of adult male mink, % females whelped, number of kits born per female, kit survival to weaning were not affected	Wren et al. 1987b
Rat *Rattus norvegicus* Strain: Sprague-Dawley	Adult	F	NR	3900	Reproductive cycle[b]	NR	No effect on maternal body weight, litter size, behaviour of dams or offspring; 4% lower bw in offspring; biochemical changes	Sundberg and Okarsson 1992

Species	**Life Stage**	**Sex**	**N**	**Daily dose $\mu g \cdot kg^{-1}$ bw**	**Duration of exposure**	**Total dose $mg \cdot kg^{-1}$ bw**	**Endpoint and Effects**	**Reference**
Rat *Rattus norvegicus* Strain: Wistar rat	Adult	F	NR	10-50	Days 6-9 of gestation	0.04 - 0.2	Decrease in operant behaviour of 4 mo old offspring; LOAEL 10 $\mu g \cdot kg^{-1}$ bw	Bornhausen et al. 1980

NR = Not Reported

Notes:

[a] 1 $mg \cdot kg^{-1}$ fed daily for 10-12 weeks, then every other day for remainder of 6 month study (daily dose caused mortality to 3; 6 animals in discomfort were euthanized).

[b] Fed to dam, 11 weeks prior to mating, during gestation and lactation; measured levels in various dam and pup tissues.

APPENDIX C-3: SUMMARY OF DATA ON THE CARCINOGENIC AND MUTAGENIC EFFECTS OF ORALLY-ADMINISTERED METHYLMERCURY IN MAMMALS.

Species	Life Stage	Sex	N	Daily dose $\mu g \cdot kg^{-1}$ bw	Duration of exposure	Total dose $mg \cdot kg^{-1}$ bw	Endpoint and Effects	Reference
Mice *Mus musculus* Strain: ICR	Adult	M	60	906 (10 ppm)	104 weeks	942	Renal epithelial tumours in 13/29 ; none at lower doses at 58 weeks	Hirano et al. 1986
Mice *Mus musculus* Strain: ICR	Adult	F	60	785 (10 ppm)	104 weeks	816	Epithelial degeneration of renal proximal tubules; degeneration of the sciatic nerve	Hirano et al. 1986
Rat *Rattus* spp. Strain: unknown	Adult	M	25	0-100 2.5 ppm	2 years	to 73	No difference in tumours among all groups	Verschuuren et al. 1976
Rat *Rattus* spp. Strain: unknown	Adult	F	25	0-100 2.5 ppm	2 years	to 73	No difference in tumours among all groups	Verschuuren et al. 1976
Rat *Rattus norvegicus* Strain: Sprague-Dawley	Adult	M	56	280	130 weeks	255	No tumours occurred.	Mitsumori 1983, 1984
Rat *Rattus norvegicus* Strain: Sprague-Dawley	Adult	F	56	280	130 weeks	255	No tumours occurred.	Mitsumori 1983, 1984
Rat *Rattus norvegicus* Strain: Sprague-Dawley	Adult	M	30	8 0.2 ppm	2 years	6	No difference in tumours between treatment and control. No other effects.	Newberne et al. 1972
Rat *Rattus norvegicus* Strain: Sprague-Dawley	Adult	F	30	8 0.2 ppm	2 years	6	No difference in tumours between treatment and control. No other effects.	Newberne et al. 1972

APPENDIX C-4: SUMMARY OF DATA ON THE ACUTE, CHRONIC OR REPRODUCTIVE TOXICITY OF METHYLMERCURY IN BIRDS.

Species	Life Stage	Sex	N	Dose $mg \cdot kg^{-1}$ in diet $\mu g \cdot kg^{-1}$ $bw \cdot d^{-1}$	Length of exposure	Total dose $mg \cdot kg^{-1}$ bw	Tissue conc. $mg \cdot kg^{-1}$ ww	Endpoint and Effects	Reference
Black duck *Anas rubripes*	Adult	M/F	26	3 (free choice)	196 d	NR	3-6 Brain ducklings	Dose fed to adult pairs; Fewer eggs laid; reduced hatch success; Reduced survival of offspring	Finley and Stendell 1978
Common loon *Gavia immer*	Adult	NR	NR	>0.3 in perch 53^{a}	Breeding season	NR	1.6 Muscle 0.5 Brain	Reduced territory establishment, reduced egg laying	Barr 1986
Common loon *Gavia immer*	Adult	NR	NR	>0.36 in perch 72	Breeding season	NR	2.3 Muscle 0.8 Brain	Reduced territory establishment, reduced egg laying, no chicks survived	Barr 1986
Double-crested cormorant	Young	NR	NR	0.56 NR	Season	NR	0.63 Brain	No effect observed.	Wolfe and Norman 1998
Double-crested cormorant	Adult	NR	NR	0.56 NR	Season	NR	NR	No effect observed.	Wolfe and Norman 1998
Great Blue heron *Ardea herodias*	Young	NR	NR	0.56 73-210	Season	NR	0.35 Brain	No effect on growth rates of young.	Wolfe and Norman 1998
Great. Blue heron *Ardea herodias*	Adult	NR	NR	0.56 122	Season	NR	NR	Reproductive success comparable to uncontaminated colonies.	Wolfe and Norman 1998
Mallard *Anas platyrhynchos*	Eggs	NR	80	NR	18 d	1-90 µg/egg	0.05-0.53 /egg	Malformations at 1 µg; decreased growth at 9 µg; 68% survival at 27 µg	Hoffman and Moore 1979

Species	**Life Stage**	**Sex**	**N**	**Dose** **mg·kg^{-1} in diet** **µg·kg^{-1} bw·d^{-1}**	**Length of exposure**	**Total dose mg·kg^{-1} bw**	**Tissue conc. mg·kg^{-1} ww**	**Endpoint and Effects**	**Reference**
Mallard *Anas platyrhynchos*	YOY	M/F	48	0.5, 5, 15 (free choice)	84 d	NR	NR	Kidney abnormalities at 5 and 15 mg·kg^{-1}	Snelgrove-Hobson et al. 1988
Mallard *Anas platyrhynchos*	Adult 1st season	F M	30 9	0 (control) 6.4^{b}	21 weeks	NR	<0.05 Eggs	Egg production ceased after 43 weeks.	Heinz 1974
Mallard *Anas platyrhynchos*	Adult 1st season	F M	30 9	0.5 dw 86^{c}	21 weeks	NR	~1 Eggs	Egg production ceased after 31 weeks. Smaller eggs than controls, ducklings hyper-responsive in avoidance tests	Heinz 1974
Mallard *Anas platyrhynchos*	Adult 1st season	F M	30 9	3.0 dw 530^{d}	21 weeks	NR	6.5-9.2 Eggs	Egg production ceased after 21 weeks. Smaller eggs, fewer sound eggs, reduced hatching success, reduced hatchling survival to 1 wk (no statistics), ducklings hyper-responsive in avoidance tests	Heinz 1974
Mallard *Anas platyrhynchos*	Adult 2nd season, 1st gen.	F M	10 10	0 (control) 6.4^{b}	1.2-1.5 years	NR	<0.05 Eggs <0.07 Liver		Heinz 1976a

Species	Life Stage	Sex	N	Dose mg·kg^{-1} in diet µg·kg^{-1} bw·d^{-1}	Length of exposure	Total dose mg·kg^{-1} bw	Tissue conc. mg·kg^{-1} ww	Endpoint and Effects	Reference
Mallard *Anas platyrhynchos*	Adult 2nd season, 1st gen.	F M	9 9	0.5 dw 83^e	1.2-1.5 years	NR	0.8-1.1 Eggs 1.6 Liver 1.8 Kidney 0.8 Breast 0.5 Brain 0.6 Ovary 11.2 Feathers	No consistent differences	Heinz 1976a
Mallard *Anas platyrhynchos*	Adult 2nd season	F M	10 10	3.0 dw 450^f	1.2-1.5 years	NR	5.5-7.4 Eggs 11.1 Liver 14.7 Kidney 5.0 Breast 4.6 Brain 8.4 Ovary 68.7 Feathers	Reduced hatchling survival to 1 wk, Brain lesions. Ducklings hyper-responsive in avoidance tests	Heinz 1976a
Mallard *Anas platyrhynchos*	Adult 2nd gen	F M	14 14	0 (control) 6.4	From 9 days of age	NR	<0.05 Eggs <0.05 Livers		Heinz 1976b

Species	Life Stage	Sex	N	Dose mg·kg^{-1} in diet µg·kg^{-1} bw·d^{-1}	Length of exposure	Total dose mg·kg^{-1} bw	Tissue conc. mg·kg^{-1} ww	Endpoint and Effects	Reference
Mallard *Anas platyrhynchos*	Adult 2nd gen	F M	14 14	0.5 dw 73^{g}	From 9 days of age	NR	0.9 Eggs 0.9 Liver 1.5 Kidney 0.7 Breast 0.4 Brain 0.5 Ovary 9.0 Feathers	Adults consumed significantly more food than controls. Increase in % eggs laid outside the nest box. Decrease in number of normal 1 wk old ducklings (but not in % normal hatchlings surviving to 1 wk). Ducklings gained less weight.	Heinz 1976b
Mallard *Anas platyrhynchos*	Adult 3rd gen.	F M	14	0 (control)	From 9 days of age	NR	<0.05 Eggs 0.1-1.4 Liver		Heinz 1979
Mallard *Anas platyrhynchos*	Adult 3rd gen	F	14	0.5 67^{h}	From 9 days of age	NR	0.86 Eggs 1.5 Liver 1.6 Kidney 0.8 Breast 0.6 Brain 0.6 Ovary 9.1 Feathers	Hens laid fewer sound eggs. Egg shells thinner. Offspring displayed impaired behaviour	Heinz 1979
Mallard *Anas platyrhynchos*	Adult	M	12	10 (4 ww) 1000	75 d	75	NR	Weak legs after 7 weeks; most barely walking and one dead at end of study.	Heinz and Hoffman 1998
Mallard *Anas platyrhynchos*	Adult	F	9	10 (4 ww) 1000	75 d	75	NR	Reduced egg hatch, reduced survival of young to 7 d, fewer young produced.	Heinz and Hoffman 1998

Species	Life Stage	Sex	N	Dose mg·kg^{-1} in diet µg·kg^{-1} bw·d^{-1}	Length of exposure	Total dose mg·kg^{-1} bw	Tissue conc. mg·kg^{-1} ww	Endpoint and Effects	Reference
Ring-necked pheasant *Phasianus colchicus*	Adult	F	7	9[i] NR	84 d	34	7.8 Liver	Weight loss; demyelination of spinal cord. Fewer eggs produced. Shell-less eggs; low hatch success. High chick mortality.	Fimreite 1971
Ring-necked pheasant *Phasianus colchicus*	Adult	F	7	4.5 NR	84 d	19	4.5 Liver	Weight loss; demyelination of spinal cord. Fewer eggs produced. Shell-less eggs; low hatch success.	Fimreite 1971

NR = Not Reported

Notes:

[a] Loons: 0.3 µg·g^{-1} x 730 g·d^{-1}·4.13 kg^{-1} bw = 53 µg·kg^{-1} bw·d^{-1} (CCME 1998).

[b] Calculated using a measured dose of 0.05 mg·kg^{-1} dw and a food ingestion rate for control mallards of 0.128 kg dry weight of food per kg body weight per day (Heinz 1974, 1979)

[c] Calculated using a measured dose of 0.55 mg·kg^{-1} dw and a food ingestion rate for treated mallards of 0.156 kg dry weight of food per kg body weight per day (Heinz 1974, 1979)

[d] Calculated using a measured dose of 3.4 mg·kg^{-1} dw and a food ingestion rate for treated mallards of 0.156 kg dry weight of food per kg body weight (Heinz 1974, 1979)

[e] Calculated using a measured dose of 0.53 mg·kg^{-1} dw and a food ingestion rate for treated mallards of 0.156 kg dry weight of food per kg body weight per day (Heinz 1974, 1979)

[f] Calculated using a measured dose of 2.88 mg·kg^{-1} dw and a food ingestion rate for treated mallards of 0.156 kg dry weight of food per kg body weight per day (Heinz 1976a, 1979)

[g] Calculated using a measured dose of 0.47 mg·kg^{-1} dw and a food ingestion rate for treated mallards of 0.156 kg dry weight of food per kg body weight per day (Heinz 1976b, 1979)

[h] Calculated using a measured dose of 0.43 mg·kg^{-1} dw and a food ingestion rate for treated mallards of 0.156 kg dry weight of food per kg body weight per day (Heinz 1976b, 1979)

[i] Fed contaminated grain because MeHg used as a fungicide on grains at the time (grains are no longer MeHg treated in Canada).

APPENDIX D: HUMAN CONSUMPTION RESTRICTION CATEGORIES FOR METHYLMERCURY IN FISH FROM CANADIAN WATERS.

Unrestricted Consumption - Fish may be consumed freely. Children under 15 and women of child-bearing age should eat fish only in this category. Fish in this category have concentrations less than or equal to **0.5 $\mu g \cdot g^{-1}$**.

Limited Consumption - Consumption restrictions depend upon the mercury level in the fish, how much fish is consumed and the length of time over which the fish is consumed.

Group #1: Fish in this group have mercury concentrations between **0.5 to 1.0 $\mu g \cdot g^{-1}$**. Short term consumption (for one week only) - anglers can eat 2300 grams (5.0 lb.) of fillets or about ten meals. Long term Consumption (fish consumed throughout the year) - anglers can eat 200 grams (0.5 lb.) of fillets or about one meal per week.

Group #2: Fish in this group have mercury concentrations between **1.0 to 1.5 $\mu g \cdot g^{-1}$**. Short term Consumption (one week only) - anglers can eat 1500 grams (3.0 lb.) of fillets or about 7 meals. Long term Consumption (Fish consumed throughout the year) - anglers can eat 140 grams (0.3 lb.) of fillets or about one small meal per week.

No Consumption- No Fish should be consumed from this category. Fish in this category have concentrations greater than **1.5 $\mu g \cdot g^{-1}$**.

Health Canada
Health Protection Branch
Bureau of Chemical Safety
1st Floor East, Banting Building
Postal Locator 2201B1
Ottawa, Ontario
KIA OL2

April 7, 1998

Our *reference* number: ADDCP98032001

Ms. Brenda Miskimmin:
Limnos Freshwater Consultants

Dear Ms. Miskimmin:

This is in reference to your fax and inquiry of March 19, 1998 regarding the 0.5 ppm guideline for mercury in fish.

This guideline was established in 1969 by the Health Protection Branch, and was based on the estimated consumption of commercial fish by eaters of that food commodity and on the available toxicology relating to mercury, and in particular, to methylmercury. Please note that this guideline relates only to fish which are marketed commercially, and that there are exemptions to this guideline. These exemptions are fresh and frozen fillets of shark, swordfish and tuna, where an advisory of limiting consumption of these species to, at most, one meal per week was recommended. The decision to exclude these species from the 0.5 ppm guideline was based on information relating to typical mercury levels in these species, and that strict application of the guideline would severely restrict the continued availability of these species in Canada to the consumer.

We would point out that there also exists a guideline of 0.2 ppm total mercury, which is recommended when fish constitutes a major subsistence food, as is the case for some aboriginal individuals in some smaller communities. This recommendation was issued by the Medical Services Branch in 1979, and reiterated in 1984.

The 0.5 ppm guideline was based on the provisional tolerable daily intake of methylmercury established by the FAO/WHO Joint Expert Committee on Food Additives (JECFA) in 1978, and reiterated in the 1988 JECFA meeting. It is listed in the 1998 edition of the **General Standard for Contaminants and Toxins in Foods** which is published by the Codex Alimentarius (copy attached).

We trust the above will be of assistance to you.

Yours truly

N.P. McEwen
Additives & Contaminants Section
Chemical Health Hazard Assessment Division

Attach.

1998

CODEX ALIMENTARIUS GENERAL STANDARD FOR CONTAMINANTS AND TOXINS IN FOODS

ANNEX IV -B SITUATION REVIEW OF CONTAMINANTS AND TOXINS IN FOODS

1.13 Mercury

(1) Mercury is a trace element which is found widespread in nature, due to volcanic and industrial activities. It has no established function in the body. Complex bound mercury (Hg) and in fish methylmercury are the predominant species. Mercury compounds are relatively toxic, especially organic bound mercury. Organic mercury compounds have been used in the past as fungicides, but are now generally forbidden for that use. Contamination of the environment with mercury compounds has been reduced in most countries by the discontinuation of agricultural uses and by adaptations in industrial processes and in waste management. In the past several tragic cases of poisonings have occurred due to mercury compounds, mostly with fish, due to environmental contamination, but also related to food use of treated cereals intended for sowing.

(2) The 1972 JECFA evaluation establishing a PTWI (tolerable weekly intake) of 5 µg/kg body weight for total mercury (out of which a maximum of 3.3 µg/kg may be methylmercury) was confirmed by the 1988 JECPA.

There are extensive analytical and intake data available, identifying fish and fishery products as the main source of mercury in the diet. Cereals have been contributing in the past too, in relation to the seed treatment with mercury compounds.

Mercury levels in plant products are usually very low. This is also valid for animal products (except fish), but higher levels might occur when fish meal is an important item of the animal feed.

The average daily intake of mercury is reported to be between a few to 20 µg, mainly varying with the amount of fishery products in the diet. For consumers with a high intake of fish, the intake may be higher. but in normal situations still considerably below the PTWI.

(3) Health problems may occur in contaminated areas and/or in relation to contaminated Products. There are indications that naturally occurring higher mercury levels in predatory marine fish are less toxic due to complexing with selenium.

(4) Many countries have national MLs, especially regarding mercury in fish, but sometimes also regarding other foods.

There is a WHO guideline value of 0.001 mg/L for total mercury in drinking water

(5) The exposure of the general population and of groups at risk may be reduced by prohibiting fishing and sale of fish from heavily contaminated waters. Advice to consumers about fish consumption may also be useful in specific situations.

(6) Codex standards exist for mercury in fish. Further action is desirable on that point regarding the definition of the residues and regarding the specification of

predatory fish. A detailed position paper is necessary as a basis for any other future action regarding possible MLs for mercury in other foods. Presently available information does not seem to justify establishing of MLS in other food commodities.